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Vol. 25 APRIL, 1949 No. 4

# SURGERY

*A Monthly Journal Devoted to the Art and Science of Surgery*

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Vol. 25, No. 4, April, 1949. Surgery is published monthly by The C. V. Mosby Company, 3207 Washington Blvd., St. Louis 3, Mo. Subscription price: United States, its possessions, Pan-American Countries, $10.00 a year; Canada and foreign, $11.00. Entered as Second-Class Matter January 20, 1937, at the Post Office at St. Louis, Missouri, under the Act of March 3, 1879. Printed in U. S. A.

# In the spastic colon

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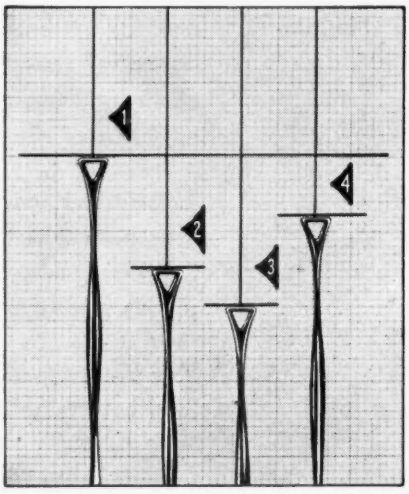


**(Fig. 1)** An 0 gauge Champion-Paré Serum-Proof Silk Suture prior to stress. **(Fig. 2)** The same, suspending a 5 lb. weight. **(Fig. 3)** The same suture, suspending a 10 lb. weight. **(Fig. 4)** After weights are removed.

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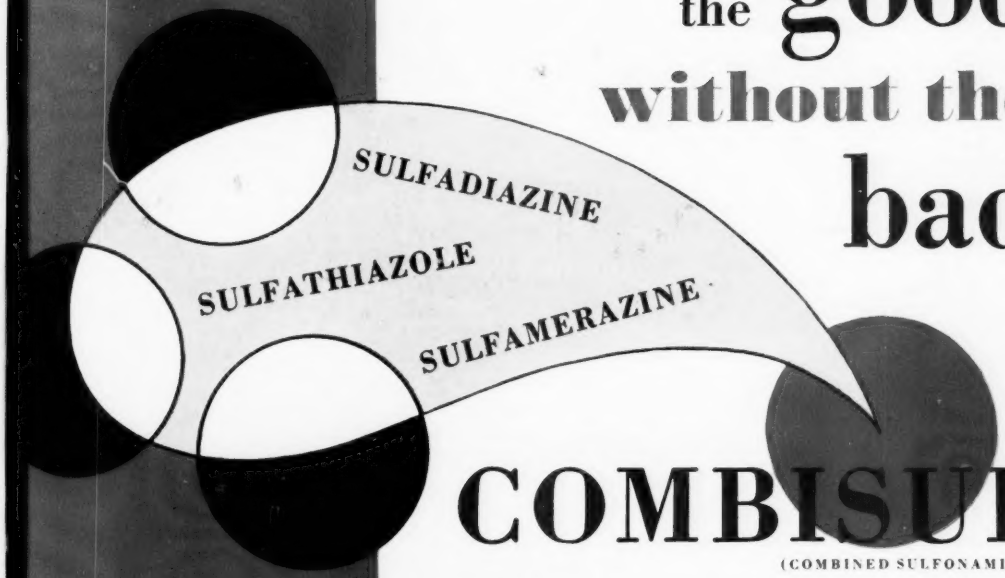


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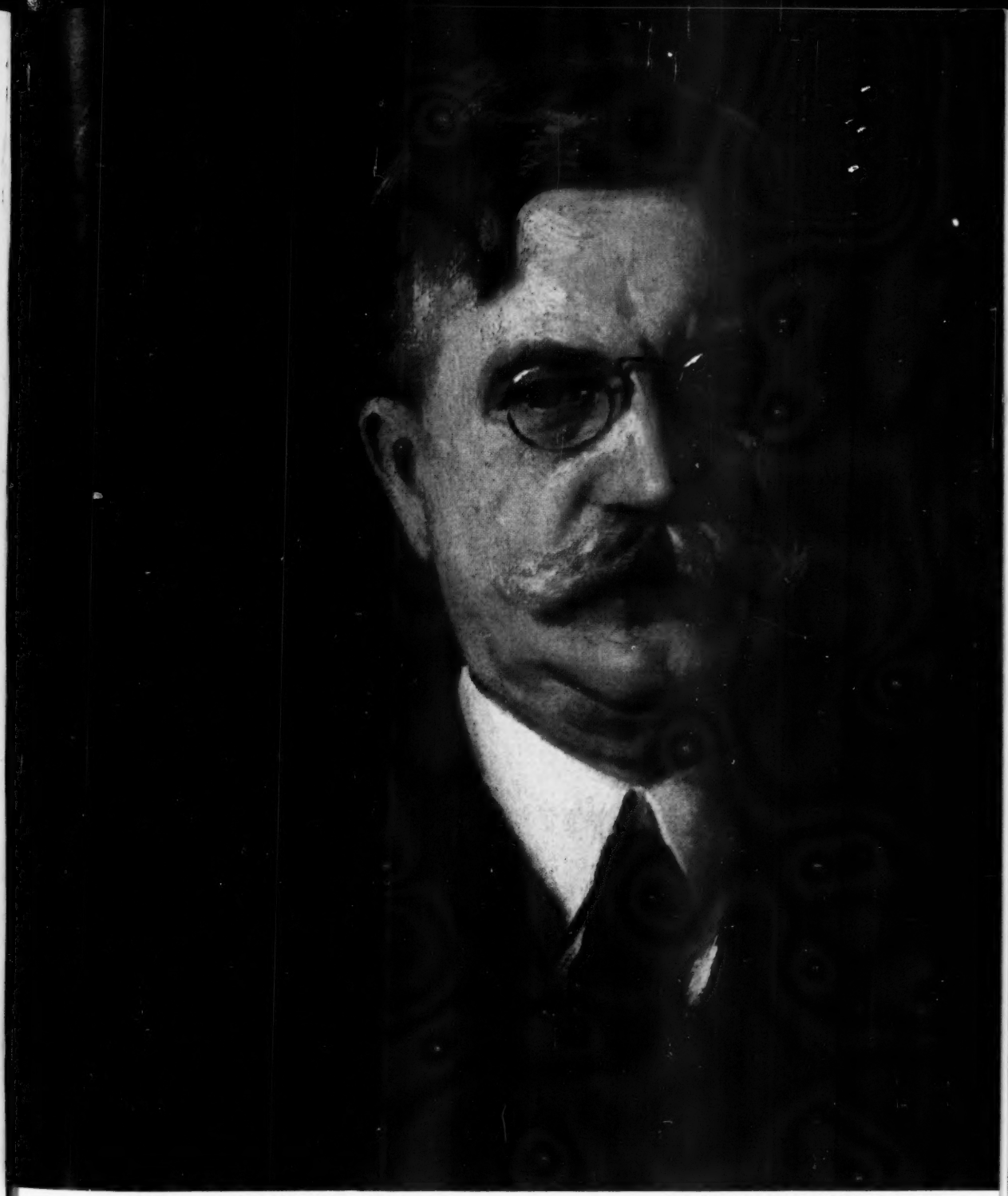

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1862-1933

Dr. Stewart received many honors in his distinguished career of nearly 44 years in the practice and teaching of surgery.

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Dr. Stewart was born in Cumberland County, Nova Scotia. In 1899 he was graduated from Bellevue Hospital Medical College (New York University). Later he was professor of surgery at N.Y.U.; in 1916, head of surgery.

One of the greatest honors Dr. Stewart received was the presentation by the late George F. Baker of $1,000,000 to New York University to be known as the George David Stewart Endowment for Surgery.

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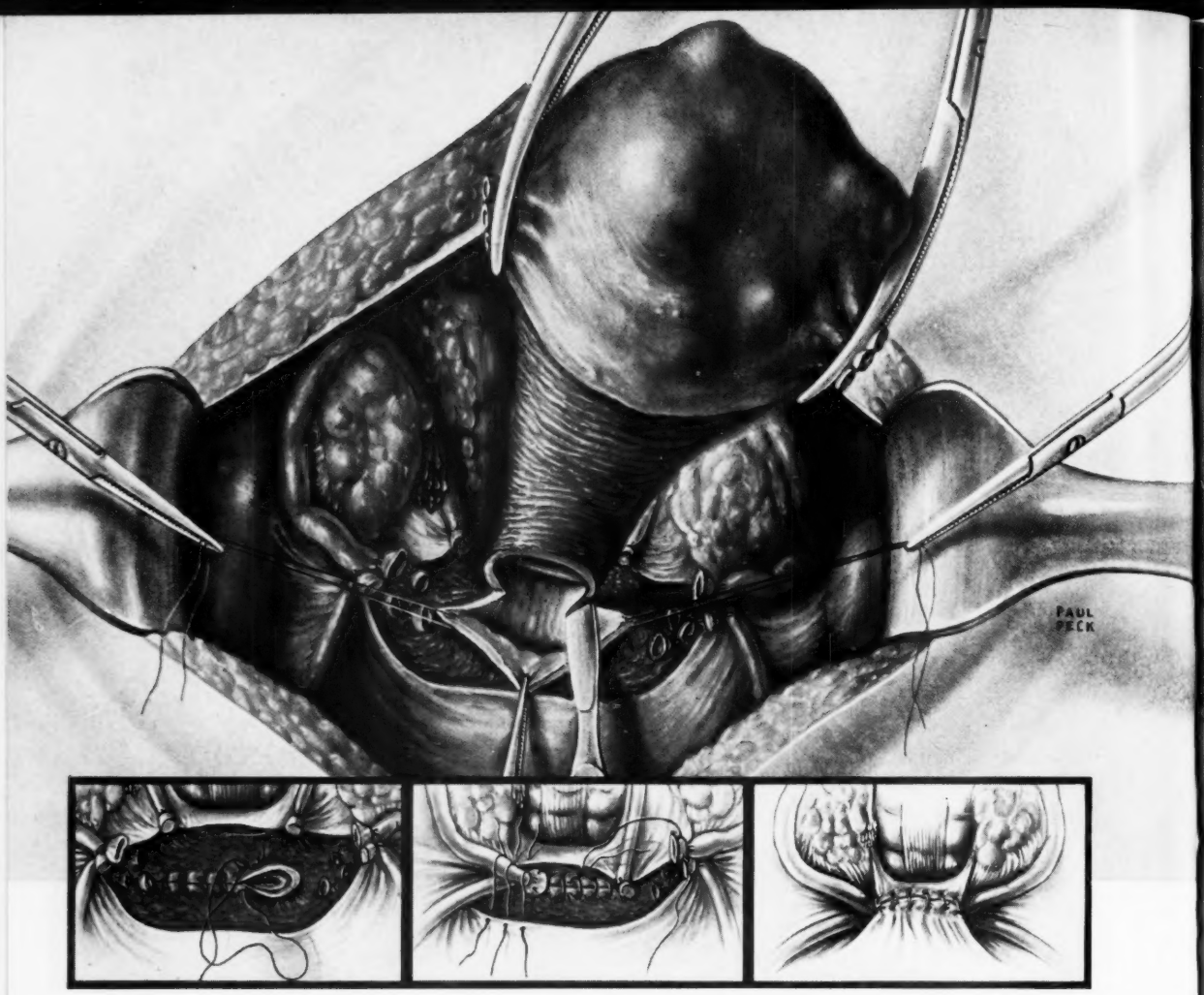



# Advances in Ob. & Gyn. Suturing

## *NEW CLINICAL PROCEDURES FAVOR NEEDLE SUTURES*

Sutures swaged to eyeless needles are being more widely adopted for use in delicate tissues by Gynecologists. Many Obstetricians have changed entirely to eyeless needles for episiotomy closure.

This trend has been revealed by Ethicon's recent six-month study in many prominent hospitals. As a result, a new group of 13 Ob. and Gyn. eyeless needle sutures has been developed. There are four new needles, swaged to chromic gut, sizes 3-0 to 2.

The clumsy bulk of the eyed needle, combined with the doubled strand, with its attendant tearing or ripping of tissue, has influenced the more meticulous technicians to take advantage of the refinements derived from the eyeless needle.

Many surgeons have objected to eyed needles, used out of necessity, which are often too large, bent, dull, or even rusted. The Atraloc eyeless needle eliminates these difficulties as well as the time wasted in rethreading needles at critical moments.

The Atraloc eyeless needle draws a single strand of suture through the tissues, eliminating confusion and minimizing trauma in the often difficult and potentially dangerous suturing encountered in Ob. and Gyn. work. It is carefully engineered and is made by highly skilled craftsmen.

All Ethicon Ob. and Gyn. eyeless needles are swaged to Ethicon's Tru-gauged, Truchromicized Surgical Gut, noted for its superior strength and flexibility.

### *Swaged Needles At No Extra Cost*

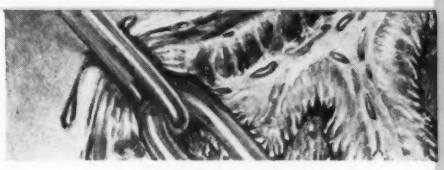
These new Ethicon sutures with swaged needles are supplied to hospitals at no extra cost over the regular Standard Tubes of Surgical Gut. They are delivered in the new, unbreakable metal Sterile Pack canisters which guarantee sterility of tube exteriors.

## Increasing Preference for Eyeless Needles

A trend toward eyeless needles is evident in Ob. & Gyn. suturing. Leading surgeons in this field prefer a non-traumatizing needle, they reported to the Ethicon Development and Survey Department.

The illustrations at the right show the contrast between the old style eyed needle and the new streamlined, eyeless Atraloc needle supplied by Ethicon.

The eyed needle does greater injury to tissue because it pulls a looped suture in its wake. The eyeless needle causes minimal trauma by pulling a single strand, slightly smaller than the needle.



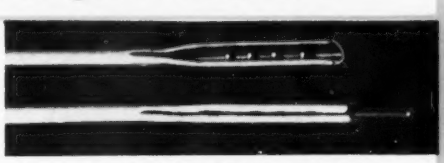
EYED NEEDLE

ATRALOC NEEDLE

## Ethicon's New Seamless Swaged Needles

The newest improvement in swaging eyeless needles is the Ethicon seamless swage, shown contrasted with the conventional swage in the illustrations at the right.

As shown, the swage has no seam. The suture is inserted in a bored aperture in high-tempered steel, then locked by compression. No rough edges or gaps to scrape tissue. This new swage is only one-quarter as long as the conventional swage, yet tests show superior holding power.



CONVENTIONAL SWAGE

ETHICON SEAMLESS SWAGE

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In addition to the seamless swage, Ethicon's new Atraloc Needles offer the surgeon these additional advances and improvements.

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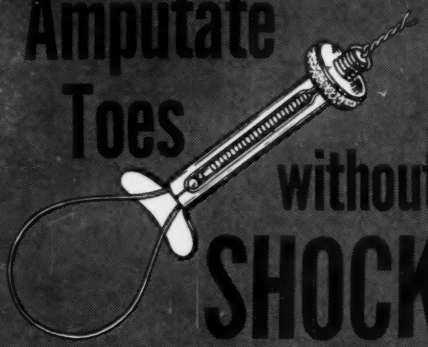

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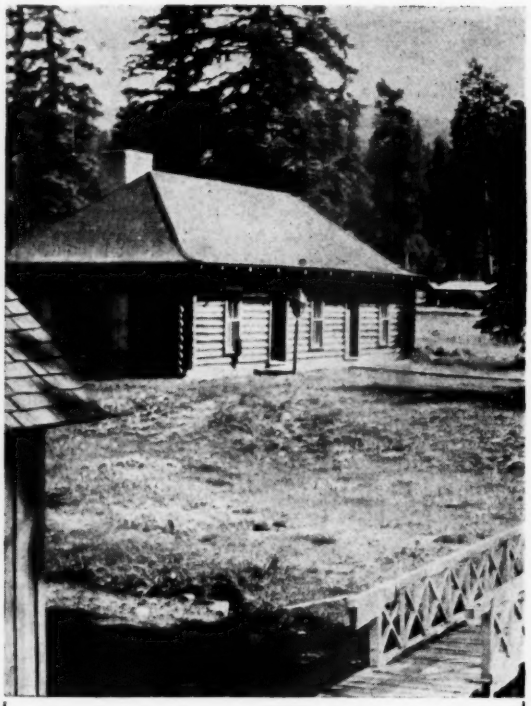

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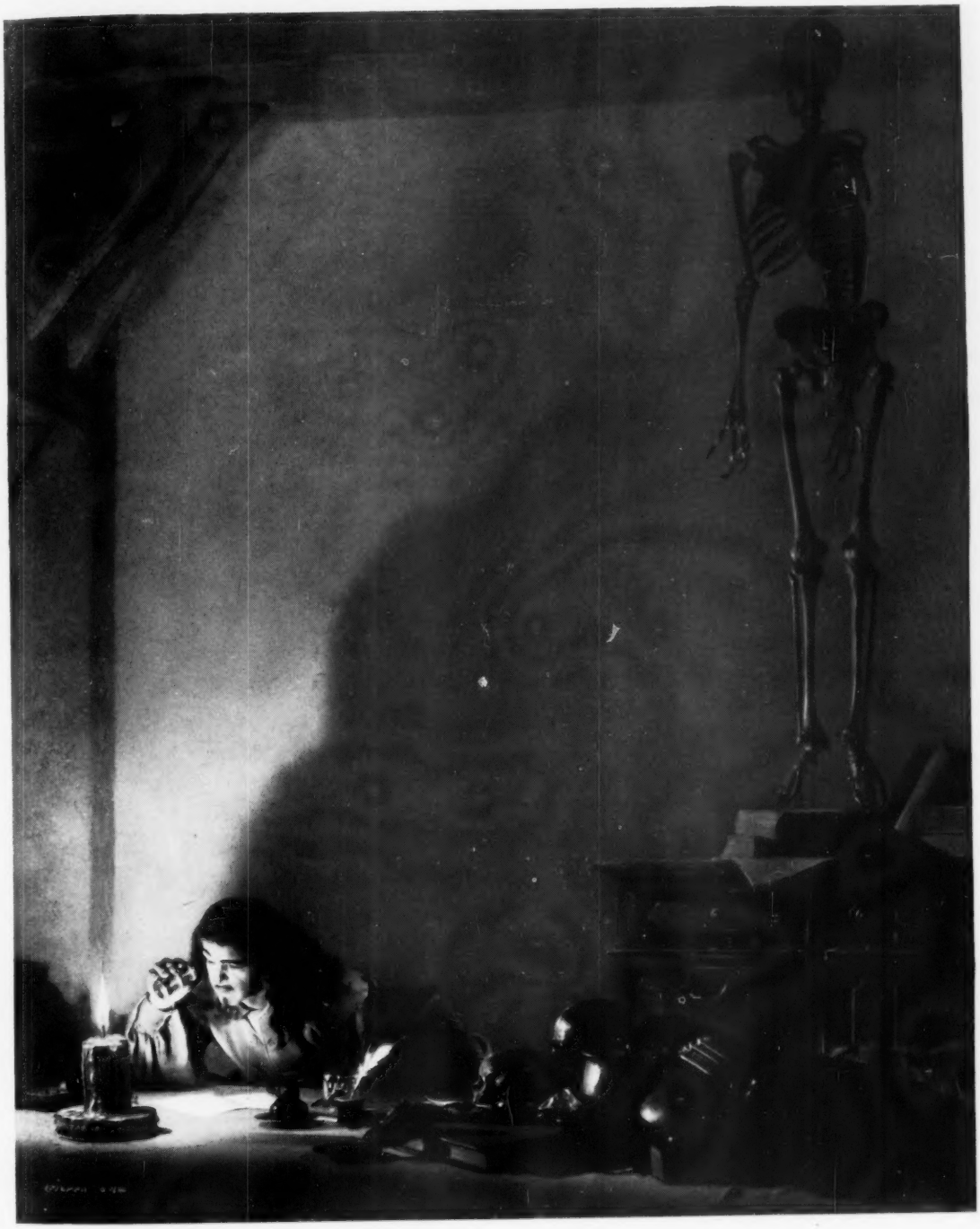


*Marcello Malpighi*

(1628-1694)

# SUTUREGRAM

NUMBER TWELVE IN A SERIES

## Suturing of Traumatic Wounds

Traumatic wounds should not be sutured primarily unless:

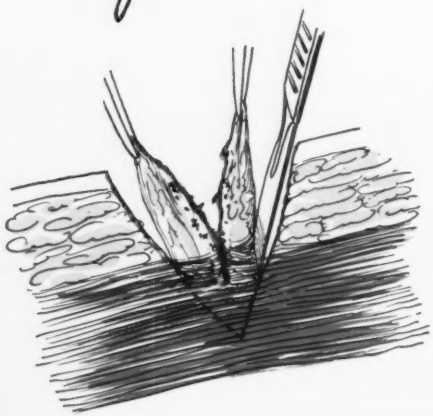
(1) Interval preceding treatment is less than ten hours

(2) All foreign bodies are removed

(3) All crushed tissue is removed

(4) Bacterial contamination is minimized

Last three factors accomplished by debridement



# SUTUREGRAM

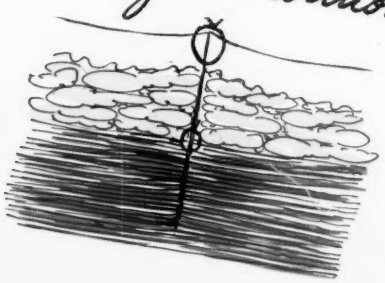
**NUMBER TWELVE (CONTINUED)**

If conditions are not obtained, then delayed primary suturing should be done

✓ Step #1 Debridement

✓ Step #2 Wound packed open with gauze

✓ Step #3 Five to eight days later gauze removed, remnants of slough excised and wound sutured in layers without drainage



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AMERICAN VOLUME
*Editor:* WILLIAM A. ROGERS, M.D.
8 The Fenway
Boston 15, Massachusetts, U. S. A.

BRITISH VOLUME
*Editor:* SIR REGINALD WATSON-JONES
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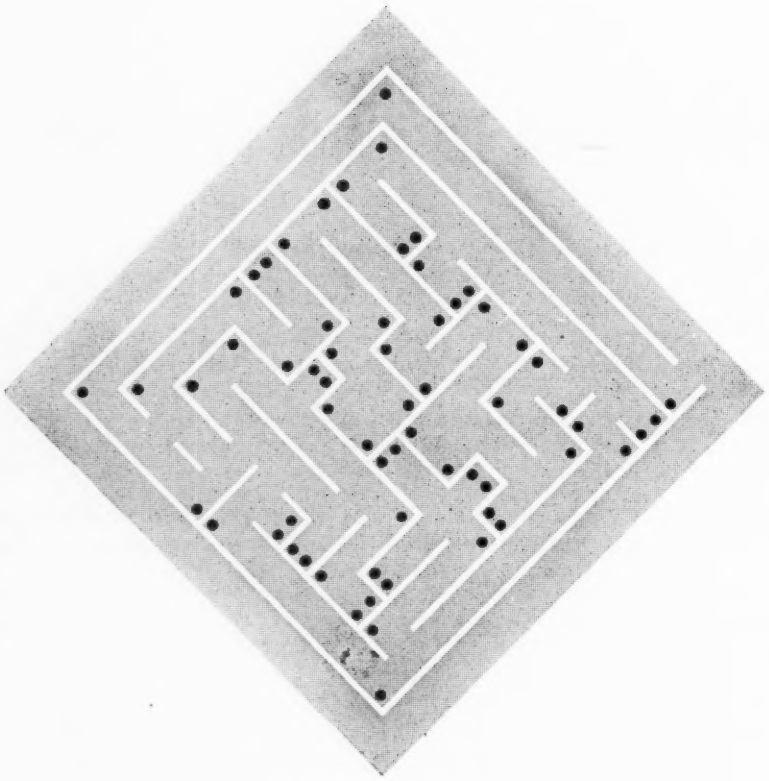

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# SURGERY

VOL. 25 APRIL, 1949 NO. 4


## Original Communications

### AN EXPERIMENTAL STUDY OF COLLATERAL CORONARY CIRCULATION PRODUCED BY CARDIOPNEUMONOPEXY


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IN VIEW of the prevalence of coronary artery disease, several intensive studies have been directed within the last decade toward devising means of producing collateral coronary circulation. Several possible sources of such a collateral blood supply exist: the thoracic wall, including the pectoral muscles, the internal mammary artery, and the intercostal arteries; the pericardium; the omentum; and the lung. Investigators in this country and abroad have undertaken experimental studies utilizing various of these sources (Fig. 1).

Interest in the surgical approach to the problem of coronary artery disease was stimulated by Claude Beck,[56] who formulated the concept of the "trigger zone" of myocardial anoxemia and of a prophylactic "blood bath" to such a functionally impaired area. He emphasized the fact that complete occlusion of all branches of a coronary artery, however small, supplying a given area of the heart resulted in far more rapid and irreversible damage to myocardial function than partial occlusion of much larger vessels.

Beck's first operation involved covering the heart with a flap of the pectoralis major muscle and suturing it to the parietal pericardium, using the vessels of that muscle as a source for a collateral blood supply.[51, 52, 53, 59, 60, 61, 80] In subsequent studies he effected a new blood supply to the heart through vascular adhesions resulting from mechanical abrasion and chemical irritants.[56, 88, 89] He also found that vascularization of adhesions produced by either of these methods or by a combination of both was adequately developed only in those animals with definite antecedent myocardial ischemia. This observation has been corroborated by Lezius and by us. In two series of 50 dogs each, Beck ligated the anterior descending branch of the left coronary artery 0.5 cm. below its bifurcation, with a mortality of 70 per cent in one series and 68 per cent in the other. With simultaneous abrasion of the entire epicardial surface, mortality from ligation was 60 per cent in another series of 50 animals. When abrasion


Received for publication, July 26, 1948.

was done from one to three weeks prior to ligation, the mortality was 38 per cent. In 19 experiments in which adhesions were produced by the introduction of finely powdered asbestos into the pericardial sac, the mortality from subsequent ligation was 31 per cent. The degree of infarction was found to be less in the three series of animals with protective adhesions than in the two series with ligation alone.[56]

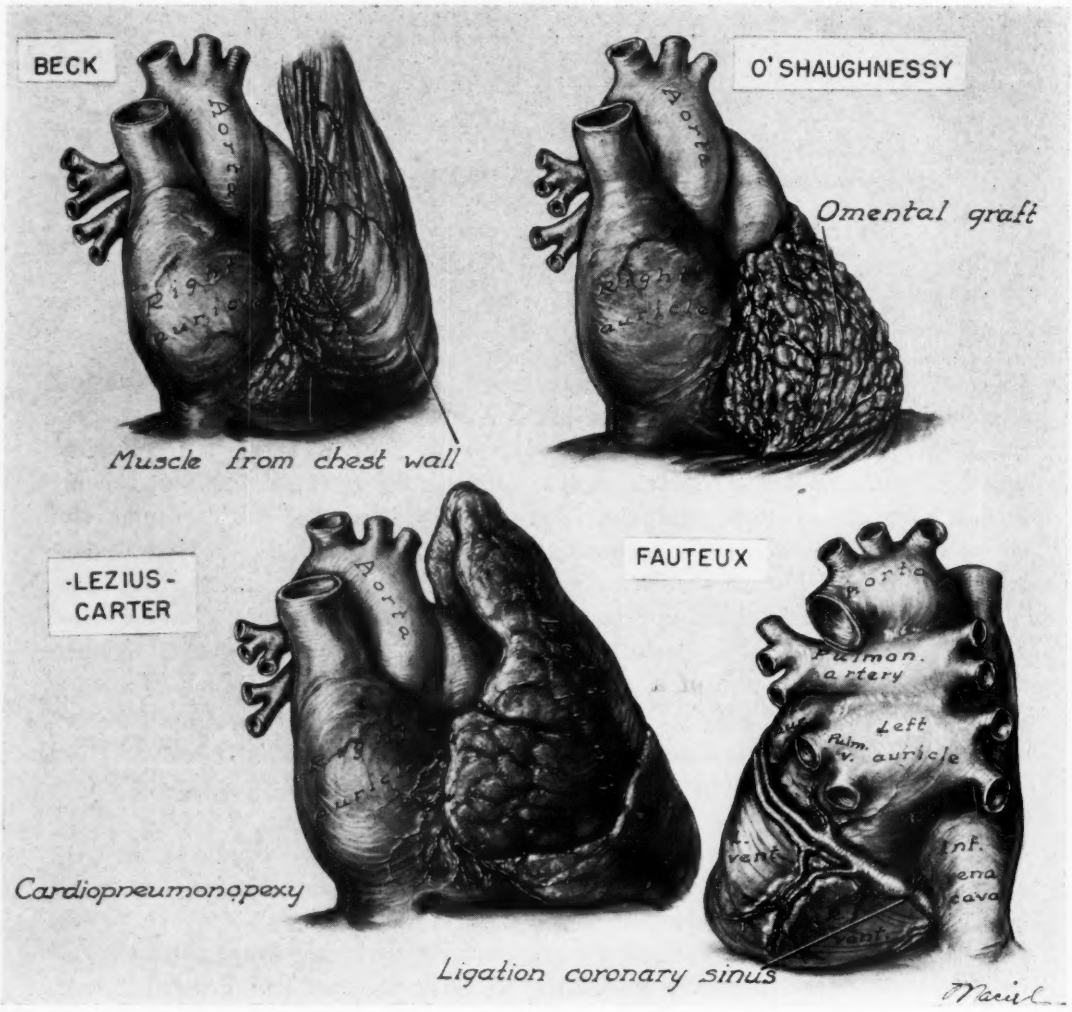


Fig. 1.—Various methods of producing collateral circulation.

O'Shaughnessy (1936-1937) [62, 84, 85, 86] used the omentum as a source for a collateral coronary blood supply, bringing it up through the diaphragm and suturing it to the surface of the heart. Although he reported no mortality statistics in his experimental studies, he described diminished exercise tolerance in cats and dogs following ligation of the left coronary artery with restoration of tolerance almost to normal following cardio-omentopexy in these same animals. He was able to demonstrate anastomoses between the omentun and the heart by

means of Thorotrast. He also performed experimental cardiopneumonopexy on 2 animals, reporting the production of demonstrable adhesions.

Meanwhile Lezius (1937-1938)[78, 79] in Germany was concerned with the utilization of the lower or middle lobe of the left lung as a source for a collateral blood supply to the heart. He produced sterile adhesions by painting the opposing surfaces of both organs with a 35 per cent solution of trypaflavine and suturing them together. He believed that cardiopneumonopexy could more adequately produce compensatory circulation than any previously described method, and that it had the added advantage of being a more easily performed operation. Without giving statistics, he reported that ligation of the left coronary artery proximal to its bifurcation was carried out following cardiopneumonopexy in 18 dogs, 2 of which died with open pneumothorax. The remaining 16 were subjected to systematic running exercises with no evidence of functional difficulty. In 3 of these, subsequent ligation of the right coronary artery was performed without untoward effect.

Heinbecker and Barton (1941)[76, 77] produced pericardial adhesions by introducing an irritating mixture of Aleuronat, gelatin, starch, glycerin, and water through a small opening in the pericardium by means of a "grease gun" with a long curved nozzle, and suturing the pericardium to the retrosternal tissue. Intercoronary communications thus produced were well developed, but the authors did not believe that sufficient protection against death from myocardial ischemia was afforded. All 14 control animals died within three to five minutes after ligation of both branches of the left coronary artery, while in 14 animals with previously induced adhesions, mortality following ligation of both branches of the left coronary artery was 57 per cent. Thompson[90, 91, 92] investigated the production of vascular pericardial adhesions by means of talc with more favorable results, reporting a series of 16 dogs with previously induced adhesions after ligation of the descending branch of the left coronary artery. In 8 animals the production of adhesions was followed by successive ligation of all coronary arteries. Two dogs survived the entire procedure. Thompson[90, 91, 92] and Heinbecker and Barton[76, 77] were the only American investigators to report ligation of more than one major coronary branch. Beck,[56] reporting the studies of both, was unable to corroborate the results of either.

Fauteux (1946),[63, 64, 65, 67, 68] although not attempting to produce collateral coronary circulation, sought to relieve the symptoms of coronary artery disease by combining coronary vein ligation with pericoronary neurectomy. In a series of 15 animals prepared by this procedure, he found that the survival rate following ligation of the circumflex branch of the left coronary artery at its origin was increased to 86.7 per cent. In his control series of 20 dogs only 20 per cent had survived similar ligation, while 60 per cent of 10 animals with coronary vein ligation alone withstood the ligation of the circumflex branch of the left coronary artery.

After consideration of the various methods reported, it was decided that the lung appeared to provide the most effective source for a collateral blood supply to the heart. The fact that Lezius' work appeared to give the best demonstrable results substantiated this belief. We therefore attempted cor-

roboration of Lezius' results, introducing certain technical modifications. Beck showed that trypaflavine (acriflavine), the substance used by Lezius, is a strong irritant and therefore not well suited for application to the epicardium. Beck[88] reported that of the many agents used producing vascular adhesions which he tested, a suspension of finely powdered asbestos in saline solution was the safest and most effective. We have accordingly selected asbestos as the irritant for the production of adhesions in the operation which we term cardiopneumonopexy. In view of the possibility of interference with coronary circulation, and the potential danger to the heart, the sutures uniting the lung to the heart, described by Lezius, were not used.

## EXPERIMENTAL STUDIES

*Technique of Cardiopneumonopexy.*—Mongrel dogs are used, the average-sized animal being given ½ gr. morphine sulfate intramuscularly one hour before operation. After intravenous injection of sodium nembutal (veterinarian), 1 gr. per five pounds weight, an intratracheal tube is passed, and the dog is placed on the right side, exposing the left chest. The intratracheal tube is attached to the windshield wiper apparatus described by Beck[55] for maintaining alternating positive oxygen pressure. The entire left chest is shaved and the skin carefully prepared by washing with soap and water for 10 minutes, ir-

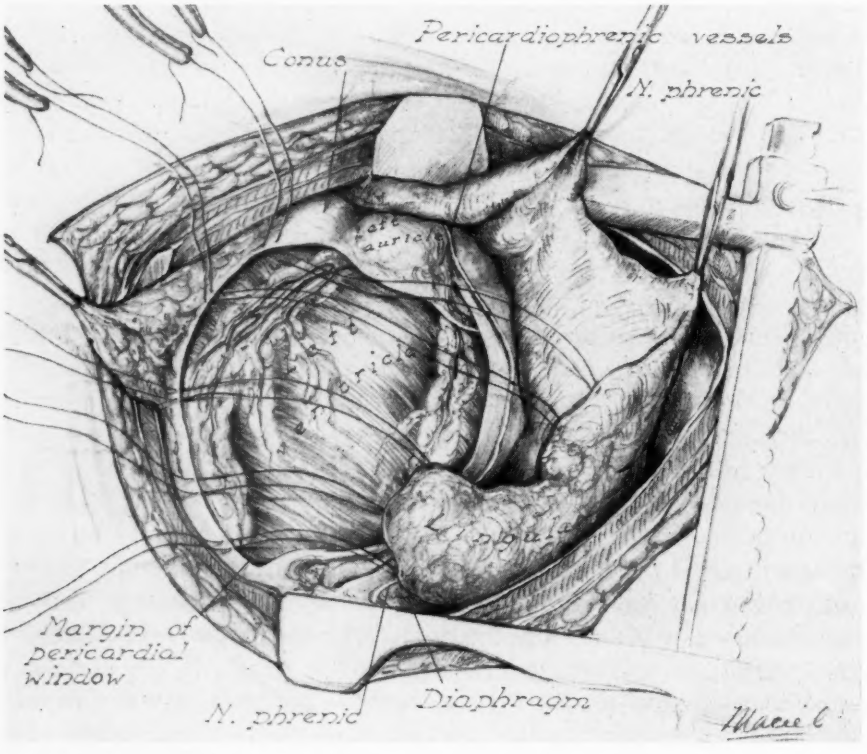


Fig. 2.—The mattress sutures of silk have been placed between the medial edge of the pericardial window and the edge of the lung. Asbestos paste will be applied to the opposing surfaces of heart and lung and the sutures pulled up and tied.

rigating with normal saline solution, and painting with 7 per cent tincture of iodine, alcohol, and ether. Sterile precautions and aseptic operative technique are maintained throughout.

A thoracotomy incision is made in the left fourth interspace, and adequate exposure is obtained without resection of costal cartilage or ribs by the insertion of a self-retaining retractor. The left phrenic nerve is identified, mobilized, and retracted, and the pericardial fat is mobilized and retracted medially. Particular attention is directed to avoid opening the right pleural cavity through the mediastinum. Five cubic centimeters of 2 per cent procaine solution is introduced into the pericardial sac through a small opening and several minutes are allowed to elapse. The opening is then prolonged superiorly to within 1 cm. of the left auricular appendage and inferiorly to within 2.5 cm. of the tip of the apex. The anterior descending branch of the left coronary artery is identified and its mid-portion used as the center for a circular "window" 4 to 5 cm. in diameter which is made in the parietal pericardium. Three or four interrupted mattress sutures are placed between the medial edge of this pericardial window and the medial border of the middle lobe of the left lung (Fig. 2). Finely divided sterile asbestos powder, mixed in sufficient saline solution to form a paste, is applied to the surface of the exposed myocardium and the opposed under surface of the left middle lobe. The mattress sutures are pulled up and tied, the lung is inflated, and closure of the incision in layers is accomplished. The operation can be performed quite easily and quickly, and is accompanied by no operative mortality except for that resulting from the complications of infection associated with operations on dogs.

This report concerns studies on 54 animals. The dogs developing infection have been excluded from consideration.

*Series I. Normal dogs with cardiopneumonectomy:* Twelve dogs were prepared by cardiopneumonopexy according to the manner just described. All animals had received medical investigation preoperatively, including weight, blood pressure, venous pressure, circulation time, chest roentgenogram, and electrocardiogram. Each of these examinations were repeated within 72 hours of sacrifice of the animal. The only significant difference between pre- and postoperative findings was the development of typical electrocardiographic evidence of pericarditis in all dogs postoperatively. After sacrifice, the heart and lungs were removed and injected to determine by gross and microscopic study whether new vascular channels could be demonstrated between the normal myocardium and the adherent lung in dogs with no functional impairment of the coronary blood supply.

*Technique of Injection.*—Under intravenous nembutal the dog is bled to death by means of a cannula placed in the left femoral artery. The blood is collected, citrated, and later mixed with a 10 per cent India ink to form a suspension for injection purposes. After the trachea has been isolated in the neck and clamped, the right thoracic cage is removed and the lobes of the right lung are excised, the hilar structures of each lobe having been individually tied with braided silk. The mediastinum is opened; the superior and inferior vena cava are ligated and divided; and the azygos vein, the innominate and left subclavian

arteries, and the thoracic aorta are doubly ligated and divided. The trachea, heart, and left lung are removed as a single specimen. By careful sharp and blunt dissection the branches of the pulmonary arteries and veins supplying the upper, middle, and lower left lobes are isolated. Cannulae are tied in place, the location being determined by the phases of circulation being studied. One cannula is connected to an injection apparatus consisting of a mercury manometer interposed between an oxygen tank and the bottle of injection fluid. Systemic pressure is maintained by means of the oxygen tank, while the pressure of the fluid in the cannula is controlled by the manometer. At the conclusion of the experiment, specimens are sent to pathology laboratory for gross and microscopic examination.

*Series Ia. Injection through the left middle lobe pulmonary artery (Table I):* In 6 dogs, sacrificed between 27 and 116 days after cardiopneumonopexy, the specimen was injected through the pulmonary artery to the left middle lobe. India ink-citrated blood suspension was used in 5 of these, and India ink in water in the other. The pulmonary veins draining the left lung were ligated in 4 and cannulated in 2. Injection was carried out for 30 minutes under pressure of 30 to 40 mm. mercury.

Grossly, there was no evidence of ink in the myocardium. Microscopically, however, all specimens in this series showed ink in several epicardial arteries and in the smaller vessels of the superficial myocardium. Occasionally major coronary arteries beneath the adherent lung showed ink, and in several specimens there was penetration into deep myocardial vessels. New vascular channels between heart and lung were uniformly demonstrated.

*Series Ib. Injection through the left middle lobe pulmonary vein (Table II):* Specimens from 3 dogs sacrificed from 18 to 29 days after cardiopneumonopexy were injected through the pulmonary vein of the adherent lobe for 30 minutes at a pressure of 10 to 40 mm. mercury. A cannula placed in the left pulmonary artery was allowed to drain. There was no gross evidence of India ink in the myocardium, but microscopic examination revealed ink in the epicardial vessels, chiefly veins, in all specimens (Fig. 3). In one specimen there were local areas of penetration of the deep myocardial vessels by injection fluid. Evidence of communication between veins of the lung and myocardium was demonstrated microscopically in normal hearts with injections carried out at pressures as low as 10 mm. mercury.

*Series Ic. Injection through the left coronary artery (Table III):* In specimens from 3 dogs sacrificed 13 to 32 days after cardiopneumonopexy, injection was accomplished through the anterior descending branch of the left coronary artery for an average of 20 minutes at pressures from 80 to 100 mm. mercury. Cannulae were inserted to drain all branches of the left pulmonary artery and veins. Grossly the India ink solution was seen to drip from the cannula in the left middle lobe pulmonary vein. Microscopic examination showed vessels of the adherent lung widely filled with injection fluid in every instance.

TABLE I. SERIES IA. CARDIOPNEUMONOPEXY ON NORMAL HEARTS; INJECTION THROUGH LEFT MIDDLE LOBE PULMONARY ARTERY

| DOG NO. | DATE OF CARDIO-PNEUMONO-PEXY | SACRIFICE DATE AND INTERVAL | INJECTION FLUID | PRESSURE AND TIME | OTHER VESSELS TREATED | GROSS OBSERVATIONS | MICROSCOPIC OBSERVATIONS |
|---|---|---|---|---|---|---|---|
| 24 | 6/18/46 | 9/30/46 103 days | Thorotrast with India ink | 40/ 0 30 min. | Pulmonary veins from left lung ligated | India ink visible in coronary bed | Several large epicardial arteries and veins contain ink; at one point arterial branches containing ink penetrate myocardium deeply |
| 26 | 6/26/46 | 10/11/46 107 days | 10% India ink in $H_2O$ | 40/ 0 30 min. | Pulmonary veins from left lung ligated | India ink visible in small branches of coronary artery at base of heart | Many newly formed arterioles filled with India ink extending into epicardium; some small arterioles and venules in epicardium filled; excellent penetration of myocardium |
| 27 | 7/ 1/46 | 10/18/46 110 days | India ink with blood | 40/ 0 30 min. | Pulmonary veins from left lung ligated | Left lower lobe well filled; no gross India ink in myocardium | Several major epicardial arteries ink-filled; occasional deep penetration into myocardium of ink |
| 5 | 7/ 3/46 | 10/25/46 115 days | India ink and blood | 40/24 31 min. | Pulmonary veins from left lung ligated | Left upper lobe and left middle lobe slightly dark; left lower lobe quite dark | Several large arteries and one major coronary artery in epicardium, and superficial myocardium contain ink; no deep penetration into myocardium |
| 28 | 7/ 5/46 | 10/28/46 116 days | India ink and blood | 30/20 40 min. | Cannulae in pulmonary veins from all left lobes | India ink dripped freely from left middle and upper lobe cannulae | Ink in several small epicardial arteries and veins; excellent ink extension into capillaries of superficial 1/3 of myocardium at apex of left ventricle |
| 29 | 10/ 8/46 | 11/4/46 27 days | India ink and blood | 40/30 30 min. | Cannulae in pulmonary veins from all left lobes | Good outflow of injection fluid from cannulae in left upper and middle lobes | Occasional large arteries and venules contain ink; many arterioles and venules in superficial myocardium contain ink |

TABLE II. SERIES Ib. CARDIOPNEUMONOPEXY ON NORMAL HEARTS; INJECTION THROUGH PULMONARY VEINS IN LEFT MIDDLE LOBE

| DOG NO. | DATE OF CARDIO-PNEUMONO-PEXY | SACRIFICE DATE AND INTERVAL | INJECTION FLUID | PRESSURE AND TIME | OTHER VESSELS TREATED | GROSS OBSERVATIONS | MICROSCOPIC OBSERVATIONS |
|---|---|---|---|---|---|---|---|
| 30 | 10/10/46 | 11/ 8/46 29 days | India ink and blood | 40/25 29 min. | Cannula in left pulmonary artery | Myocardium appeared filled with ink; good flow from pulmonary artery | Several epicardial veins contain ink, and there is extension via veins and capillaries 3 mm. into myocardium |
| 36 | 10/24/46 | 11/11/46 18 days | India ink and blood | 10/ 5 30 min. | Cannula in left pulmonary artery; left upper lobe pulmonary vein also injected | Questionable obstruction in cannula in left middle lobe pulmonary vein | Several epicardial veins contain ink; no artery filled, no penetration into myocardium |
| 37 | 10/25/46 | 11/22/46 28 days | India ink and blood | 20/ 0 20 min. | Cannula in anterior descending branch of left coronary and left pulmonary artery | 30 c.c. of the injection fluid collected from cannula in left pulmonary artery; no other cannulae drained at all | Many epicardial arteries and veins contain ink; several areas of deep myocardial penetration by ink-filled arteries and veins |

TABLE III. SERIES Ic. CARDIOPNEUMONOPEXY ON NORMAL HEARTS; INJECTION THROUGH LEFT CORONARY ARTERY

| DOG NO. | DATE OF CARDIO-PNEUMONO-PEXY | SACRIFICE DATE AND INTERVAL | INJECTION FLUID | PRESSURE AND TIME | OTHER VESSELS TREATED | GROSS OBSERVATIONS | MICROSCOPIC OBSERVATIONS |
|---|---|---|---|---|---|---|---|
| 38 | 10/29/46 | 11/11/46 (death) 13 days | India ink and blood | 80 /0 15 min. | Cannulae in left pulmonary artery and all branches of left pulmonary veins | India ink dripped slowly from left middle lobe vein cannulae only | |
| 31 | 10/14/46 | 11/15/46 32 days | India ink and blood | 100/0 20 min. | Cannulae in left pulmonary artery and all branches of left pulmonary veins | Cannulae in left pulmonary artery and pulmonary veins of left middle lobe dripped injection fluid quickly | Gross filling of pulmonary arteries and veins |
| 34 | 10/17/46 | 11/18/46 32 days | India ink and blood | 80 /0 20 min. | Cannulae in left pulmonary artery and all branches of left pulmonary veins | Injection fluid dripped from cannulae in left middle lobe pulmonary vein and pulmonary artery; 45 c.c. collected from vein and 5 c.c. from artery | Gross filling of pulmonary arteries and veins |

*Series II:* For comparison with the results obtained by cardiopneumonopexy on the normal heart, a second group of studies was performed to determine its effect on the ischemic myocardium. In a control group of 23 dogs, simple ligation of the anterior descending branch of the left coronary artery was performed. In a second group of 19 dogs, ligation followed cardiopneumonopexy at an interval of 14 to 154 days. The two groups were contrasted from the standpoint of mortality and survival rate and of gross and microscopic appearance of the infarcts in the survivors. In addition, animals surviving ligation subsequent to lung transplant were sacrificed and the injected specimens compared with those in Series I.

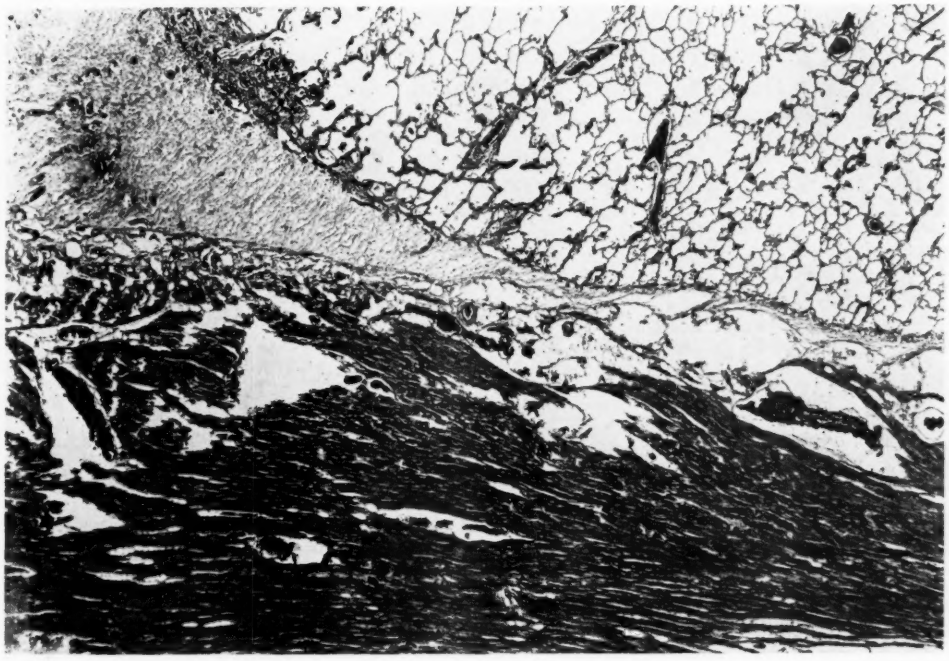

Fig. 3.—Photomicrograph showing injection of the superficial vessels in a normal heart after cardiopneumonopexy. The adherent lung is shown at the top with intervening granulation tissue between it and the myocardium below.

*Technique of Ligation of the Anterior Descending Branch of the Left Coronary Artery.*—Under intravenous nembutal with positive oxygen pressure a thoracotomy incision is made in the fourth left interspace. The pericardial fat and the left phrenic nerve are mobilized and retracted out of danger. Five cubic centimeters of 2 per cent procaine solution are instilled into the pericardial sac through a small opening in the parietal pericardium, and 1 minute is allowed to elapse. The opening is then extended longitudinally directly over the anterior descending branch of the left coronary artery. A figure-of-eight traction suture is placed in the tip of the left auricle to permit its lateral retraction in order to expose the origin of the left coronary artery. The anterior descending branch is carefully isolated by sharp and blunt dissection and at a point 0.5 cm. distal to its junction with the circumflex branch it is ligated in continuity or doubly ligated and divided. The pericardium is closed with interrupted sutures of fine silk and layer closure of the chest is accomplished.

*Series IIa. Control series with simple left coronary artery ligation (Table IV):* In 23 animals operated upon according to the technique just described, the survival rate was 52 per cent and the mortality rate was 48 per cent. In Beck's larger series with ligation of the descending branch of the left coronary artery, the mortality was about 70 per cent. The low mortality rate of 48 per cent in our series may be attributable to the liberal use of procaine and the high degree of oxygenation maintained prior to, during, and after ligation. Death occurred within 20 minutes after ligation in 8 cases, usually as a result of left ventricular fibrillation with visible cyanosis and acute dilatation of the area made ischemic. Three dogs lived 8, 17, and 96 hours, respectively, after operation. Necropsy was performed on all dogs not surviving the procedure to verify the point of placement of the ligature. No dog was included in the series unless the septal and circumflex branches were patent and the cause of death was acute infarction or its sequelae.

TABLE IV. SERIES IIA. LIGATION OF ANTERIOR DESCENDING BRANCH OF LEFT CORONARY ARTERY

| DOG NO. | LIGATION | SURVIVAL | SACRIFICE | NECROPSY FINDINGS |
|---|---|---|---|---|
| 61 | 12/13/46 | Yes | 5/21/47 | Ligature correct; large infarct anterior apex |
| 64 | 12/30/46 | Yes | 5/19/47 | Ligature correct; large infarct anterior apex |
| 66 | 12/27/46 | Yes | 5/19/47 | Ligature correct; large infarct anterior apex |
| 68 | 1/20/47 | Yes | 5/21/47 | Ligature correct; medium-sized infarct posterior apex |
| 70 | 1/21/47 | No | ---- | Died in ventricular fibrillation in 17 min. |
| 72 | 1/16/47 | No | ---- | Died in ventricular fibrillation in 20 min. |
| 78 | 1/27/47 | No | ---- | Died in ventricular fibrillation in 7 min. |
| 83 | 2/ 3/47 | No | ---- | Died 17 hr. after ligation of myocardial ischemia; early apical infarct |
| 85 | 2/ 5/47 | No | ---- | Died in ventricular fibrillation in 20 min. |
| 86 | 2/ 6/47 | No | ---- | Died in ventricular fibrillation in 20 min. |
| 87 | 2/ 6/47 | No | ---- | Died in ventricular fibrillation in 10 min. |
| 95 | 2/17/47 | Yes | 5/26/47 | Ligature correct; apical scar 2.5 cm. in diameter |
| 97 | 2/17/47 | Yes | 5/19/47 | Ligature correct; large infarct left anterior apex |
| 99 | 1/28/47 | No | ---- | Died in ventricular fibrillation in 4 min. |
| 100 | 2/21/47 | No | ---- | Died on 4th postoperative day; no infection<br>Ligature correct; infarct 6 cm. in diameter at left ventricular apex |
| 103 | 2/24/47 | Yes | 5/21/47 | Ligature correct; scar 2.5 cm. in diameter on anterior apex |
| 108 | 2/28/47 | Yes | 5/26/47 | Ligature correct; anterior apical infarct 2 cm. in diameter |
| 109 | 2/28/47 | Yes | 5/26/47 | Ligature correct; anterior apical infarct |
| 140 | 4/ 4/47 | No | ---- | Died 8 hours postoperatively of acute myocardial ischemia; ligature correct |
| 141 | 4/ 4/47 | Yes | 5/28/47 | Ligature correct; large infarct left anterior apex |
| 156 | 5/20/47 | Yes | 6/16/47 | Ligature correct; no gross infarct, minimal scar ring microscopically |
| 157 | 5/21/47 | No | ---- | Died in ventricular fibrillation in 10 min. |
| 158 | 5/20/47 | Yes | 6/16/47 | Ligature correct; small infarct left ventricular apex |

The surviving animals in the control series all gave electrocardiographic evidence of acute myocardial infarction. These dogs were sacrificed 25 to 161 days after operation and the specimens were sent to the pathology laboratory for gross and microscopic examination.

*Series IIb. Left coronary artery ligation following protective cardiopneumonopexy (Table V):* Cardiopneumonopexy was performed on 19 dogs, followed at intervals of 14 to 154 days by ligation of the anterior descending branch of the left coronary artery. Both procedures followed the techniques described. In all but 3 instances at least 30 days were permitted to elapse between cardiopneumonopexy and ligation. The survival rate following ligation in this "protected" series was 78.9 per cent, and the mortality rate was 21.1 per cent. The survival rate in the protected series is thus 26.9 per cent greater than that in the control series. According to the method of Campbell,[103] to be statistically significant, the difference between any two series of figures must be more than twice the margin of possible error due to chance. In series as

TABLE V. SERIES IIB. CARDIOPNEUMONOPEXY FOLLOWED BY LIGATION OF ANTERIOR DESCENDING BRANCH OF LEFT CORONARY ARTERY

| DOG NO. | 1ST STAGE | 2ND STAGE AND INTERVAL | SURVIVAL | SACRIFICED | NECROPSY FINDINGS |
|---|---|---|---|---|---|
| 39 | 10/30/46 | 11/25/46 26 days | No | Died 17½ hr. postligation | Microscopic evidence of early infarction |
| 45 | 11/13/46 | 1/13/47 61 days | Yes | 3/28/47 | Old organizing infarct with myocardial thinning |
| 47 | 11/13/46 | 12/16/46 33 days | No | Died 15 min. postligation | Ventricular fibrillation; microscopic evidence of myocardial ischemia |
| 48 | 11/21/46 | 1/ 6/47 46 days | Yes | 5/28/47 | Anterior apical infarction; marked scarring microscopically |
| 50 | 11/27/46 | 2/ 3/47 68 days | Yes | 5/30/47 | Anterior apical infarction; marked scarring microscopically |
| 54 | 12/ 4/46 | 1/31/47 58 days | Yes | 6/ 2/47 | Large anterior apical infarct; partial scarring microscopically |
| 58 | 12/11/46 | 5/14/47 154 days | Yes | 6/25/47 | Anterior apical scar; myocardium patchily scarred microscopically |
| 90 | 2/12/47 | 3/26/47 42 days | Yes | 6/ 2/47 | Anterior apical scar; scant deep-seated scarring microscopically |
| 92 | 2/12/47 | 3/27/47 43 days | Yes | 6/ 3/47 | Anterior apex almost wholly scarred microscopically |
| 93 | 2/11/47 | 3/24/47 41 days | Yes | 6/ 3/47 | Anterior apical infarct |
| 113 | 3/ 6/47 | 5/22/47 77 days | Yes | 5/23/48 | — |
| 114 | 3/ 7/47 | 5/ 7/47 61 days | Yes | 6/ 5/47 | Left anterior apical infarct |
| 133 | 3/26/47 | 4/19/47 14 days | Yes | 6/ 4/47 | Anterior apex scantily scarred microscopically |
| 134 | 4/ 1/47 | 5/13/47 42 days | Yes | 6/17/47 | Sharply circumscribed apical infarct |
| 139 | 4/ 4/47 | 5/13/47 39 days | Yes | 6/25/47 | Thin dense apical infarct |
| 142 | 4/ 4/47 | 5/ 8/47 34 days | No | Died 20 min. postligation | Died in acute ventricular fibrillation |
| 143 | 4/ 4/47 | 5/ 9/47 35 days | No | Died 5 min. postligation | Died in acute ventricular fibrillation |
| 145 | 4/15/47 | 5/12/47 27 days | Yes | 6/ 6/47 | Bulbous anterior apical infarct |
| 148 | 4/22/47 | 5/22/47 30 days | Yes | Died 9/ 3/47 | No report |

short as these reported, the margin of error is 14, and therefore the difference between the two series fails by 1.1 per cent of being statistically significant. The figures thus seem to indicate the beneficial effect of cardiopneumonopexy on survival following ligation of the anterior descending branch of the left coronary artery though they do not statistically establish this point.

Dogs surviving the two-stage procedure were sacrificed at intervals of 22 to 142 days after ligation. Specimens from these animals were contrasted with those from the control series as to gross and microscopic appearance of the infarcts produced by ligation with and without protective transplant. These specimens were injected as in Series I, and the development of the collateral circulation under conditions of myocardial ischemia was compared with that demonstrated in normal hearts to which collaterals had been produced.

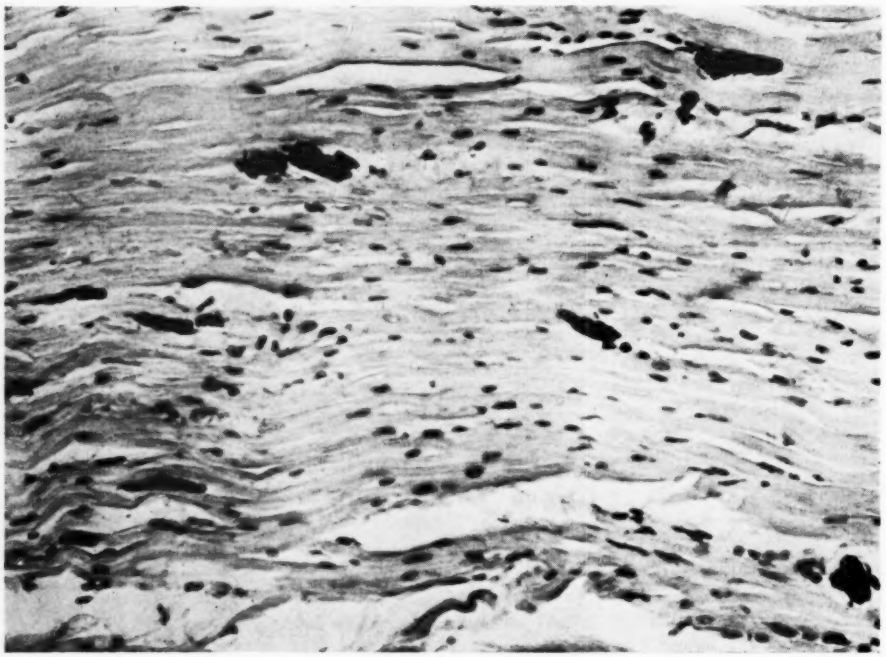

Fig. 4.—Photomicrograph showing injection of capillaries deep in the myocardium beneath the lung graft in a dog subjected to ligation of the coronary artery after protective cardiopneumonopexy.

In seven dogs surviving the two-stage operation the left middle lobe pulmonary artery was injected with India ink-blood suspension at a pressure of 40 mm. mercury for 30 minutes (Table VI). Gross filling of the major coronary vessels adjacent to the adherent lung and in the region of the apical infarcts was noted. Injection fluid drained freely from the open cannula in the left middle lobe pulmonary vein in each instance. Microscopically, good filling was obtained only in the myocardium supplied by the ligated vessel (Fig. 4). Where the muscle was intact little or no filling took place. Where revascularization occurred in the area of the scar the artery was found patent below the point of occlusion, and the collaterals were not strikingly evident.

TABLE VI. SERIES IIB. CARDIOPNEUMONOPEXY FOLLOWED BY LIGATION; INJECTION THROUGH LEFT MIDDLE LOBE PULMONARY ARTERY

| DOG NO. | DATE OF 1ST STAGE | DATE OF 2ND STAGE | DATE OF SACRIFICE | MICROSCOPIC OBSERVATIONS |
|---|---|---|---|---|
| 48 | 11/22/46 | 1/ 6/47 | 5/28/47 | Striking filling at apex of major epicardial arteries, venules, capillaries, and arterioles; abundant vascularization under adherent lung; good capillary filling of anterior surface of left ventricle |
| 50 | 11/27/46 | 2/ 3/47 | 5/30/47 | One large artery in scar containing ink, also adjacent arterioles; good filling but not much penetration at point of ligature; fair filling of posterior left ventricle |
| 54 | 12/ 4/46 | 1/31/47 | 6/ 2/47 | Several large arteries containing ink with some penetration into apical scar; penetration below adherent lung of moderate order; large artery at level of ligature occluded with recanalization |
| 58 | 12/11/46 | 5/14/47 | 6/25/47 | Grossly much ink in all vessels throughout heart; microscopically little evidence of penetration |
| 90 | 2/12/47 | 3/26/47 | 6/ 2/47 | Artery at level of ligature recanalized; occasional evidences of capillary filling, but artery patent below ligature |
| 92 | 2/12/47 | 3/27/47 | 6/ 3/47 | At anterior apex one large venule and several epicardial venules contain ink; rest of musculature intact and without filling or scarring |
| 134 | 4/ 1/47 | 5/13/47 | 6/17/47 | Injection in situ not satisfactory |
| 139 | 4/ 4/47 | 5/13/47 | 6/25/47 | Good filling but no deep penetration in area of infarct; several arteries included in scar recanalized; no dye found in intact areas of heart |

Comment: All injections except in Dog 134 (in situ) were carried out at a pressure of 40 mm. Hg.

Four other survivors of this series were sacrificed and the specimens were injected at a pressure of 10 mm. mercury for 30 minutes through a cannula placed in the left middle lobe pulmonary vein (Table VII). Not much injection fluid drained from the open cannulae in the branches of the left pulmonary

TABLE VII. SERIES IIB. CARDIOPNEUMONOPEXY FOLLOWED BY LIGATION; INJECTION THROUGH LEFT MIDDLE LOBE PULMONARY VEIN (10 MM. HG)

| DOG NO. | DATE OF 1ST STAGE | DATE OF 2ND STAGE | DATE OF SACRIFICE | MICROSCOPIC OBSERVATIONS |
|---|---|---|---|---|
| 93 | 2/11/47 | 3/24/47 | 6/3/47 | Ink present spottily in venous sinuses and arterioles both in infarct and in adjacent area; only rare venules in intact area filled; considerable venous and capillary filling in region of ligature with some recanalization |
| 114 | 3/ 7/47 | 5/ 7/47 | 6/5/47 | Moderate numbers of small venules and large sinuses near endocardium contain ink |
| 133 | 3/26/47 | 4/ 9/47 | 6/4/47 | Little scarring present, with rare filling of vessels; no dye in intact portion of the heart |
| 145 | 4/15/47 | 5/12/47 | 6/6/47 | One large artery and vein in epicardium containing ink, but no penetration; artery patent below tie, no filling; several arteries in the epicardial fat contain dye |

artery, but even at this low pressure gross filling of the major coronary arteries in the region of the apex and of the circumflex branch of the left coronary artery was noted. In two instances India ink was visible in the right coronary artery. Microscopically, dye was found only in infarcted areas, with only rare filling of vessels in intact musculature. The relation of myocardial requirements to the degree of filling was clearly seen. Comparison of the demonstrable vascular connections resulting from cardiopneumonopexy on normal hearts with those developed to hearts in which myocardial ischemia had been induced by coronary ligation further emphasized a striking development of collateral channels on the basis of need (Figs. 3 and 4).

*Myocardial Infarcts.*—Specimens from 12 control animals (Series IIa) were studied both grossly and microscopically and were compared with specimens from 13 animals in which ligation of the left coronary artery had been performed following cardiopneumonopexy (Series IIb). Complete destruction of the entire thickness of the myocardium at either the apex, interventricular septum, or both, or complete replacement at the apex and partial but extensive scarring of the interventricular septum, was regarded as indicative of severe damage; scarring extensive in area but only partial in its involvement of the thickness of the musculature was regarded as representative of moderate damage; while slight scarring at the apex with no scarring of the interventricular septum, or only superficial scarring, was regarded as evidence of minimal damage. With these criteria, the following was established: in the control series of 12 animals surviving simple ligation of the anterior descending branch of the left coronary artery (Series IIa), 9 dogs, or 75 per cent, had severe damage. In 5 dogs there was complete fibrous replacement at both the apex and interventricular septum; in 3 there was dense fibrosis of the apex and considerable scarring of the interventricular septum; and in 1 there was considerable scarring of the interventricular septum and complete cicatrization of the apex. In the remaining

TABLE VIII. CONTROL SERIES—TWELVE SURVIVORS OF LIGATION OF ANTERIOR DESCENDING BRANCH OF LEFT CORONARY ARTERY

| DOG NO. | DATE OF LIGATION | DATE OF SACRIFICE | MICROSCOPIC EXAMINATION | |
|---|---|---|---|---|
| | | | AT APEX | MIDPOINT INTERVENTRICULAR SEPTUM |
| 61 | 12/13/46 | 5/21/47 | 4 plus | Plus-minus |
| 64 | 12/30/46 | 5/19/47 | 4 plus | Plus-minus |
| 66 | 12/27/46 | 5/19/47 | 4 plus | Plus-minus |
| 68 | 1/20/47 | 5/21/47 | plus | 0 |
| 95 | 2/17/47 | 5/26/47 | 4 plus | Plus |
| 97 | 2/17/47 | 5/19/47 | 4 plus | Plus |
| 103 | 2/24/47 | 5/21/47 | 3 plus | Plus |
| 108 | 2/28/47 | 5/26/47 | 4 plus | Plus |
| 109 | 2/28/47 | 5/26/47 | 2 plus | Plus-minus |
| 141 | 4/ 4/47 | 5/28/47 | 4 plus | Plus |
| 156 | 5/20/47 | 6/16/47 | plus | 0 |
| 158 | 5/21/47 | 6/16/47 | 4 plus | Plus |

Comment: The criteria in grading the microscopic extent of the lesion refer to thickness of the infarct, and are as follows: minus, very thin scar, minimal in extent; plus-minus, moderate involvement of musculature; plus, well-defined but circumscribed scarring; 2 and 3 plus, considerable involvement; 4 plus, complete obsolescence of musculature.

3 animals (25 per cent), there was moderate to minimal damage. One dog showed partial scarring of both apex and interventricular septum, and 2 showed light scarring at the apex only.

In the "protected" series (IIb) of 15 dogs surviving ligation of the anterior descending branch of the left coronary artery following cardiopneumonopexy, 13 were sacrificed for microscopic study of the extent of infarction. Of these animals only 3, or 23 per cent, had severe myocardial damage. In one dog the apex showed a dense obsolete scar, in another the interventricular septum was completely fibrosed and the apex severely scarred, and in the third, both apex and interventricular septum showed incomplete scarring. In the remaining 10 dogs (76.9 per cent), there was moderate to minimal damage. One animal showed spotty scarring of the apex only, 1 had focal scarring of the apex with moderate scarring of the interventricular septum, 2 revealed slight scarring at both areas, 2 showed slight scarring at the apex only, and 1 showed very superficial scarring of both apex and interventricular septum.

TABLE IX. CARDIOPNEUMONOPEXY FOLLOWED BY LIGATION OF ANTERIOR DESCENDING BRANCH OF LEFT CORONARY ARTERY; STUDIES ON THIRTEEN OF SURVIVORS

| DOG NO. | DATE OF CARDIO-PNEUMONO-PEXY | DATE OF LIGATION | DATE OF SACRIFICE | MICROSCOPIC STUDY OF INFARCT | |
|---|---|---|---|---|---|
| | | | | AT APEX | AT MIDPOINT INTER-VENTRICULAR SEPTUM |
| 45 | 11/13/46 | 1/13/47 | 3/28/47 | 2 plus | 0 |
| 48 | 11/22/46 | 1/ 6/47 | 5/28/47 | 3 plus | 0 |
| 50 | 11/27/46 | 2/ 3/47 | 5/29/47 | 4 plus | 0 |
| 54 | 12/ 4/46 | 1/31/47 | 6/ 2/47 | 2 plus | Plus |
| 58 | 11/11/46 | 5/14/47 | 6/25/47 | 2 plus | Plus-minus |
| 90 | 2/12/47 | 3/26/47 | 6/ 2/47 | plus | Plus-minus |
| 92 | 2/12/47 | 3/27/47 | 6/ 3/47 | 3 plus | 0 |
| 93 | 2/11/47 | 3/24/47 | 6/ 3/47 | 3 plus | 0 |
| 114 | 3/ 7/47 | 5/ 7/47 | 6/ 5/47 | 3 plus | Plus |
| 133 | 2/26/47 | 4/19/47 | 6/ 4/47 | 2 plus | 0 |
| 134 | 4/ 1/47 | 5/13/47 | 6/17/47 | 3 plus | Plus-minus |
| 139 | 4/ 1/47 | 5/13/47 | 6/25/47 | 3 plus | 0 |
| 145 | 4/15/47 | 5/12/47 | 6/ 6/47 | 2 plus | Plus-minus |

Comment: The criteria in grading microscopic extent of the lesion are as noted on Table VIII.

A comparison of series IIa with IIb demonstrates that in addition to the strikingly lowered mortality from ligation, the severity of infarction was lessened in those animals in which protective cardiopneumonopexy had been performed. Seventy-five per cent of the dogs with ligation alone suffered severe myocardial damage, while in the protected series only 23 per cent had extensive infarction. These observations indicate the beneficial effects of cardiopneumonopexy in diminution of the severity of lesions caused by subsequent coronary occlusions.

## DISCUSSION

The results of the experiments just described clearly indicate that vascular communications between the heart and the adherent lung can be produced by

cardiopneumonopexy. They also show that the survival rate following ligation of the anterior descending branch of the left coronary artery is significantly improved following this operation and that the degree of myocardial damage is lessened. There are, however, several important physiologic questions which should be answered and which are concerned with the practical applicability of the procedure. These are chiefly three in number. (1) What is the direction of the blood flow? Is it from the lung to the heart or vice versa? (2) How great a volume of blood can be delivered through the newly established channels and how long will such channels continue to function? and (3) What is the degree of oxygenation of the blood supplied through the collateral vessels arising from the pulmonary circulation?

The direction of collateral blood flow in a given area is determined by differential pressures within the two sets of vessels involved. In the situation with which we are concerned the normal pressure in the coronary circulation is higher than that in the pulmonary, and consequently under normal conditions the flow should be from the heart to the lung rather than in reverse, which is what one would hope to accomplish by cardiopneumonopexy. Several studies have proved that normal pressures in the coronary arteries are higher than those in any other vessels. Recent work by Burwell and Dexter[23] indicates that in the normal heart the systolic pressure in the right ventricle is essentially identical with that in the pulmonary artery. Wiggers[17] found that after coronary occlusion, however, pressure curves become lower and change in contour, favoring a continuous flow from extracoronary vessels throughout the cardiac cycle, but particularly during diastole. Löhner[9] found that hearts caused to beat by perfusion through the pulmonary artery received a blood flow adequate for restricted maintenance of coronary activity. Lezius believed the principle of "blood hunger" is of the utmost importance in determining the direction of blood flow. At intervals after the establishment of cardiopneumonopexy he injected the pulmonary artery of the adherent lobe with India ink and severed the aorta after three or four heartbeats. After sacrifice of the animal large particles of ink were found in the myocardial capillaries and in the right heart, demonstrating that three to four heartbeats sufficed to pump pulmonary blood into the coronary circulation. Our own in vivo experiments with fluorescein, radioactive iodine, and India ink injections through the left middle lobe pulmonary artery proved inconclusive. Logically, myocardial ischemia together with reduced coronary artery pressure should induce flow *to* the myocardium through the anastomosis rather than the reverse, and Wiggers' experiments make clear the fact that such a process is physiologically possible. Our injection experiments following coronary artery ligation preceded by cardiopneumonopexy tend to bear out this fact.

We have not been able to establish the amount of blood which is delivered to the myocardium through the newly formed collateral channels nor have we as yet determined the period during which such channels remain patent and functional. Several animals are being maintained over a period of months or years in an attempt to clarify this point.

Since the pulmonary artery is used as the source of blood, the question of adequate oxygenation of the blood thus provided arises. Although pulmonary artery blood is usually thought to be venous in nature, several factors with regard to its nutrient value should be considered. Batson and Bellet[29] pointed out that the oxygen tension of venous blood ranges from 60 to 70 per cent, as compared with a tension of 95 per cent for arterial blood. They concluded that venous blood can nourish the ischemic myocardium to a certain extent. More important still are the recent catheterization studies of Burwell and Dexter,[23] who found that blood withdrawn from the pulmonary artery at a level as deep as the lumen of the artery permitted passage of a catheter was representative of arterial rather than venous blood. It is reasonable to suppose that the oxygenation is even greater at the still lower level from which the anastomotic vessels derive their blood supply.

In an effort to demonstrate that the collateral circulation developed from the lung after cardiopneumonopexy was functionally adequate, Lezius ligated the bronchus to the adherent lobe in an animal with a full functional recovery from ligation of the main stem of the left coronary artery following cardiopneumonopexy. The animal died within 20 hours in acute coronary insufficiency. Lezius also performed cardiopneumonopexy on an animal simultaneously with ligation of the bronchus and 6 weeks later ligated the main stem of the left coronary artery. The animal died within 11 hours. We have not thus far repeated these interesting and critical experiments.

Yet another value which may be derived from cardiopneumonopexy is indicated by the work of Murray.[83] He found that in experimentally produced cardiac infarction the infarcted area became dilated and functionally ineffectual from the time of arterial occlusion. By controlling dilatation he was able to save the lives of many of his experimental animals either by excision of the dilated area followed by immediate suture of the heart or by reinforcing the area of potential infarction with a patch of silk cloth before producing occlusion. We believe that in the case of cardiopneumonopexy the lung may afford splinting of the potentially infarcted area in a manner similar to that obtained by Murray with his second method. In addition to the protection against death from cardiac insufficiency caused by dilatation, cardiopneumonopexy affords a supplementary blood supply to the area isolated by arterial occlusion.

## SUMMARY

1. A technique for cardiopneumonopexy has been described in which asbestos powder was used as a means of producing vascular adhesions between the heart and lung.

2. Gross and microscopic evidence has been obtained that by this operation new vascular channels can be produced between the myocardium and the adherent lung, but neither the amount of blood flow, the direction of the flow, nor the duration of the patency of these channels has been established.

3. In the instances in which cardiopneumonopexy was performed on normal hearts, injections of 10 per cent suspension of India ink in blood at normal pressures revealed some filling of the superficial vessels in the myocardium, but

in the case of hearts rendered ischemic by coronary artery ligation there was considerably greater filling, not only of the superficial vessels, but of the deep ones as well.

4. Comparison of specimens obtained from dogs in which ligation of the anterior descending branch of the left coronary artery had been performed after cardiopneumonopexy with those from dogs in which ligation alone was done revealed less extensive infarcts and a lower mortality in the protected series. Whereas the mortality in the protected series was 20 per cent, in those animals with simple ligation it was 48 per cent. The frequency of severe infarction occurred in 75 per cent of animals with simple ligation, but in only 23 per cent in animals with ligation subsequent to cardiopneumonopexy.

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# THE DETERMINATION OF THE BLOOD IODINE

## A Useful Method for the Clinical Laboratory

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THE requirements of an adequate micromethod for the determination of the blood iodine, satisfactory for use in the clinical laboratory, are several: accuracy, dependability, the small amount of blood necessary, together with minimal requirements of technical ability, time, laboratory space, and expense. These requirements have been met by devising a method which requires no transfer of material during the entire procedure; and which uses small quantities of blood, small quantities of readily available reagents, and standard laboratory equipment, with the exception of an especial, readily constructed apparatus (Fig. 1).

Through using this method routinely it has been found that a trained technician can complete an analysis in less than three hours and eight or ten analyses in an eight-hour day. A minimum of five feet of laboratory-desk working space is required in addition to the use of the hood, centrifuge, water bath, and colorimeter. By limiting the directions for the method to terms of quantities, times, and temperatures, and by reducing the number of manipulations, the factors of individual skill and judgment have been reduced to a minimum. Nevertheless, to be able to introduce this method into the usual clinical laboratory, set it up, make the necessary reagents, and prepare the required standards, a technician *must* have had previous chemical experience.

### REAGENTS AND EQUIPMENT

1. *Hood.*

2. *Centrifuge.*—International centrifuge, Size 2, with attachments for centrifuging 250 ml. cups. High speed attachments such as duralumin cups and a welded steel guard are desirable, but not necessary, since the separation can be accomplished below 2000 r.p.m.

3. *Colorimeter.*—Evelyn photoelectric colorimeter* with a 440 millimicron filter. *Only uniform colorimeter tubes are used,* the rest discarded.

4. *Glassware.*—Especially an iodine analysis still (Fig. 1).

5. *Somogyi Reagent.*—

*Solution I:* 12.5 Gm. $ZnSO_4 \cdot 7\ H_2O$ C.P. is dissolved in distilled water and 125 ml. 0.25 N. $H_2SO_4$ added and this solution then diluted to one liter.

*Solution II*: 0.75 N. Na OH C.P. (this *must* be iodine-free).

Aided by a grant from The Comly Fund for Research of the Ohio State University.
Received for publication, Jan. 24, 1949.

*Rubicon Co., Philadelphia, Pa.

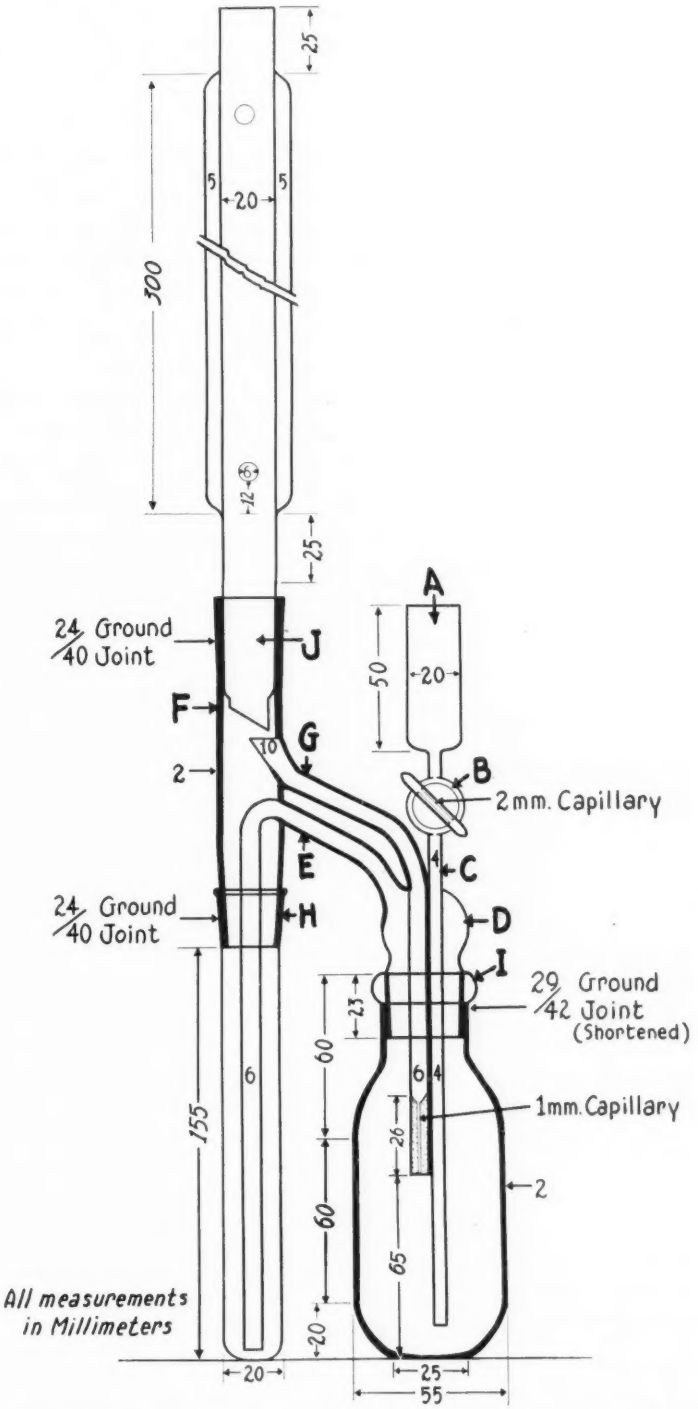


Fig. 1.—Distillation apparatus for blood iodine determination in the clinical laboratory. The distillation apparatus consists of a reagent chamber (*A*), a stopcock (*B*), a reagent tube (*C*), a distillation head (*D*), a delivery tube (*E*), a connecting tube (*F*), and a return tube (*G*). This is all in one piece and has three ground-glass joints: (*H*) at which a colorimeter tube is attached, (*I*) at which a reagent flask is attached, and (*J*) for the attachment of a water condenser.

The two solutions should be adjusted so that 6.7 ml. of Solution II is required to titrate 50 ml. Solution I to a faint pink, using phenolphthalein as an indicator.

6. *Na OH.*—Na OH pellets C. P. (Baker's). These *must* be tested for iodine before use, since an occasional bottle has an appreciable iodine content.

7. *Chromic Acid Solution.*—Prepared by dissolving 2000 Gm. $CrO_3$ (chromic acid flake, *technical grade**), in 1200 ml. distilled water.

8. *70% $H_2SO_4$.*—5 ml. concentrated HCl, C.P. is added to 500 ml. $H_2SO_4$ C.P., and the resulting solution is boiled for two hours to obtain iodine-free concentrated sulfuric acid.[1] After cooling, this is diluted with distilled water to make a 70 per cent solution, by volume.

9. *50% Phosphorous Acid Solution.*—Prepared by dissolving one bottle (453.6 Gm.) of phosphorous acid crystals† in distilled water and bringing the volume to 907 c.c.‡

10. *1% KOH.*—1.0 Gm. KOH sticks§ per 100 ml. distilled water.

11. *0.2 N Arsenious Acid.*—9.894 Gm. $As_2O_3$ C.P. is dissolved in 75 ml. 1 N. Na OH solution. Warming hastens solution. Dilute to about 300 ml. and titrate solution with dilute iodine-free $H_2SO_4$ until it is just acid to litmus. Add 90.5 ml. iodine-free $H_2SO_4$. Cool and dilute to one liter. This solution is then 3.25 N. in $H_2SO_4$.

12. *2.4 N HCl.*—Concentrated (12 N) HCl C.P. is diluted with four volumes distilled water.

13. *0.1 N. Ceric Sulfate.*—Place 58.5 Gm. ceric ammonium sulfate crystals, reagent grade‖ in distilled water. Add 103 ml. concentrated $H_2SO_4$ and dilute to about 700 ml. and allow to stand over night to dissolve. Dilute to one liter. This solution is then 3.7 N. in $H_2SO_4$.

14. *Water Bath.*—A constant temperature water bath set at 29° C.

15. *Distilled Water.*—Single distilled water has been found satisfactory in this laboratory.

16. *Hot Plate.*—660 watt, exposed-coil hot plate.

17. *KI Solution.*—Used for making standards. KI crystals C.P., dissolve and dilute to 10 micrograms iodine per milliliter. Store in a dark bottle. This will remain stable for months. Dilute further to 1 microgram iodine per milliliter for making standards. This will remain stable for weeks.

## PROCEDURE

All glassware is washed with soap and warm water immediately before using. It is then rinsed six times with tap water and twice with distilled water. Periodically, cleaning solution is used on all glassware.

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*Graselli Chemicals, Wilmington, Del.

†General Chemical Co., New York, N. Y.

‡Each lot of phosphorous acid purchased *must* be tested for its potency to release iodine. In addition, each lot *must* be tested for iodine content. Some have an appreciable iodine content. This may be reduced by adding twice the calculated amount of water and boiling down to the intended 50 per cent solution, but not beyond. Care must be taken to avoid decomposition of the phosphorous acid.

§City Chemical Corp., New York, N. Y.

‖G. Frederick Smith Chemical Co., Columbus, Ohio.

About 9 c.c. of blood are drawn from the patient, under basal conditions. This is transferred to a Wassermann tube and placed in the refrigerator for about twenty-four hours to allow for adequate clot retraction and serum separation. The clot is packed down by centrifugation. Three milliliters of serum are then pipetted volumetrically into the special 250 ml. reagent flask (Fig. 1, *I*).

Precipitation of the proteins along with the protein-bound iodine is accomplished by use of the Somogyi reagents.[2, 3, 4] Twenty-four milliliters of Somogyi Solution I are added to the flask followed by 3 ml. of Somogyi Solution II. The mixture is agitated slightly and centrifuged at 1,500 r.p.m. for five minutes. The supernatant portion, which averages about 15 ml., is poured off into a small container. This is the protein-free fraction and contains the "unbound" iodine of the blood. A 5 ml. aliquot is pipetted into an Evelyn colorimeter tube which is handled the same as the distillate. The distillate is collected later. The precipitate remaining in the reagent flask contains the physiologically significant protein-bound iodine.[5] Twenty milliliters of distilled water are added to the precipitate, which is stirred and centrifuged again. This supernatant fluid is discarded.

The precipitate is then oxidized by a mixture of chromic and sulfuric acids.[1, 6-12] Three milliliters of chromic acid solution are pipetted into the reagent flask, followed by 25 ml. of 70 per cent sulfuric acid. A two-hole rubber stopper is placed in the flask. The small hole accommodates a thermometer which extends well down into the mixture; the large hole allows for the escape of vapors. The flask with the thermometer is attached to a ring stand which has a hot plate mounted below. This assembly is placed in a hood.* The flask is heated rapidly to 218° C. and then raised off the hot plate and its temperature adjusted to about 210° C. by varying the distance between the flask and the hot plate. This temperature is maintained for about seven minutes. The flask is then allowed to cool to below 70° C. Then 15 ml. distilled water are added. The temperature is raised to 185° C. and the flask is again allowed to cool to below 70° C. This is repeated once more to insure the removal of all volatile substances.

Reduction of this solution is accomplished by phosphorous acid.[7] Thirty milliliters of distilled water are added and the flask attached to the distillation apparatus as illustrated in the accompanying diagram (Fig. 1, *I*). One milliliter of 1 per cent KOH is pipetted into an Evelyn colorimeter tube which has a ground-glass lining at the top. This tube is attached to the distillation apparatus and becomes the distillate receiver (Fig. 1, *II*). It is held in place with a burette clamp. The distillation apparatus is mounted on a tall ring stand as is also a hot plate. The hot plate is raised beneath the reagent flask and the contents brought to boiling. The hot plate is then turned off. After the solution stops boiling, the steam starts to condense and the green-colored solution rises

*An alternate procedure, and the one preferred in this clinic, is the use of a small stream of compressed air through each flask, to blow off the volatile products developed during oxidation. The exhaust may be led to a hood, out a window, or down a sink drain. If compressed air is employed the short-bulb type of thermometer should be used, so that the solution covers the bulb completely.

in the reagent tube (Fig. 1, *C*). At this time 5 ml. of 50 per cent phosphorous acid are added from the reagent chamber (Fig. 1, *A*) above, and the hot plate turned on. The addition should take 20 seconds, and is immediately followed by 2 ml. of distilled water. Distillation is continued for ten minutes. At the end of this time the hot plate is turned off and lowered, the stopcock to the reagent chamber opened, and the distillate receiver-tube lowered. The distillation stem is washed down once with a fine spray. Following this the colorimeter tube is disconnected from the apparatus, corked, and placed in a water bath set at 29° C.

At this point the iodine of the protein-free filtrate is in one Evelyn colorimeter tube and the iodine from the protein precipitate in another. Both are now treated in the same manner. The content of the marked tubes is diluted to 9 ml. using a fine spray.* The tubes are returned to the water bath. The iodine content is determined by measuring its catalytic effect on the reaction between ceric sulfate and arsenious acid.[10-17] By means of microburettes leading from reservoirs 0.5 ml. 0.2 N. arsenious acid, 0.26 ml. 2.4 N. HCl, and 0.5 ml. 0.1 N. ceric sulfate are added at thirty-second intervals. The time of addition of the ceric sulfate is noted and colorimeter readings are made each minute *on the minute* for five minutes. The colorimeter is adjusted so that the blank, a tube containing 10 ml. of distilled water, gives a reading of 100 on the lower scale. The readings taken at five minutes are used to determine the iodine content. However, if the reaction has proceeded rapidly one may use the readings obtained after a shorter interval of time.

The iodine content is determined by comparison with readings previously obtained from tubes containing known amounts of iodine. These are prepared by adding definite quantities of iodide solution, made up to contain 1 microgram of iodine per milliliter. A microburette leading from a reservoir is used to deliver the iodide solution. It is diluted to 9 ml., arsenious acid, hydrochloric acid, and ceric sulfate are added and readings are made as described previously. Graphs or tables may be prepared from this data and used as standards for comparison. A new set of standards must be prepared each time a new stock of ceric sulfate and arsenious acid is made. Also, a reagent blank must be determined each time a new set of reagents is prepared. This reagent blank is subtracted from the value found for the protein-bound iodine. Values encountered for the reagent blanks in our laboratory have ranged around 0.1 microgram of iodine.

This procedure may be carried out in any laboratory that does not have tincture of iodine, iodoacetic acid, iodine crystals, or other forms of elemental iodine open to the air. Aside from this air-borne iodine, contamination is a greatly limited possibility. Washing the hands and glassware thoroughly before making an analysis and avoiding reaching over an open flask will eliminate

*A 9 ml. mark has been placed on a card which is fixed to a sturdy test tube rack. Each colorimeter tube has its own place in the rack and its own previously determined mark. This procedure is accurate to 0.1 ml. In addition, if the dilution is short of 9 ml. the yellow color will be more intense but the iodide ion will be in higher concentration. Therefore the blanching of color takes place more rapidly. Conversely, if the dilution is past 9 ml. the color is less intense but the iodide ion is in lower concentration. In this condition the color blanches slower. If the error is less than 0.5 ml. these effects counterbalance at five minutes.

iodine contamination for all practical purposes. The other ions interfering with the ceric sulfate-arsenious acid reaction are not likely to be introduced into the solutions. These are osmium, mercury, silver, thiocyanate, cyanide, and citrate.[10] Chlorides and bromides are weak catalysts. Fluorides stop the reaction. An excess of chlorides slows the reaction. In addition, any oxidizing or reducing agent will interfere.[14] In the temperature range around 29° C., a difference of 1° C. influences the reaction velocity by about 3 per cent. At higher temperatures the reaction velocity is increased much more than this by an increase of 1° C.

In the range of iodine concentrations encountered in analyzing 3 ml. of serum, a difference of one colorimeter unit is equal to about 5 per cent of the total result. Recovery experiments using beef serum plus 10 per cent whole blood show about 100 per cent recovery plus or minus 5 per cent (Table I). An error of over 10 per cent is infrequent. Analyses done in series show an average deviation of about plus or minus 7.5 per cent for the nonprotein-bound iodine, which is determined directly (Tables II and III). However, the nonprotein-bound iodine may be determined more accurately than this if increment rather than actual colorimeter reading is used to determine the value. Also, if an abundant layer of chromic anhydride is not on the surface of the sample at the start of the analysis, recovery of added iodide will be low. This does not apply to the determination of the protein-bound iodine for which this method is used clinically.

We have found that the range of normal for the protein-bound iodine of the serum is from 4 to 6 micrograms per cent, the average being 4.8. For the

TABLE I. RECOVERY OF IODIDE ADDED TO 3 ML. SAMPLES TAKEN FROM THE SAME POOL OF SERUM

| ADDITIONS IN MICROGRAM $I^-$ | CALCULATED VALUE IN MICROGRAM $I^-$ | VALUE OBTAINED | PER CENT DEVIATION |
|---|---|---|---|
| 0 | | .31 | + 3.3 |
| 0 | | .31 | + 3.3 |
| 0 | | .29 | - 3.3 |
| 0 | | .28 | - 6.7 |
| 0 | | .28 | - 6.7 |
| 0 | | .31 | + 3.3 |
| | | Average .30 | Average deviation 4.4 |
| .1 | .40 | .39 | - 2.5 |
| .1 | .40 | .40 | 0 |
| .1 | .40 | .44 | +10.0 |
| .1 | .40 | .45 | +12.5 |
| | | Average .42 | Average deviation 6.2 |
| .2 | .50 | .50 | 0 |
| .2 | .50 | .50 | 0 |
| .2 | .50 | .45 | -10 |
| .2 | .50 | .44 | -12 |
| | | Average .47 | Average deviation 5.5 |
| .3 | .60 | .63 | + 5.0 |
| .3 | .60 | .60 | 0 |
| .3 | .60 | .57 | - 5.0 |
| .3 | .60 | .58 | - 3.3 |
| | | Average .59 | Average deviation 3.3 |
| | | | Total average deviation of additions 5.0 |

TABLE II. 25 CONSECUTIVE ANALYSES OF THE SAME SPECIMEN OF POOLED SERUM USING 3 ML.

| VALUE OBTAINED IN MICROGRAM I⁻ | PER CENT DEVIATION FROM AVERAGE |
|---|---|
| .20 | + 7.0* |
| .18 | - 3.7 |
| .17 | - 9.1* |
| .20 | + 7.0* |
| .18 | - 3.7 |
| .19 | + 1.6 |
| .18 | - 3.7 |
| .21 | +12.3* |
| .19 | + 1.6 |
| .18 | - 3.7 |
| .18 | - 3.7 |
| .17 | - 9.1* |
| .18 | - 3.7 |
| .19 | + 1.6 |
| .19 | + 1.6 |
| .19 | + 1.6 |
| .19 | + 1.6 |
| .20 | + 7.0* |
| .20 | + 7.0* |
| .18 | - 3.7 |
| .20 | + 7.0* |
| .19 | + 1.6 |
| .18 | - 3.7 |
| .18 | - 3.7 |
| .18 | - 3.7 |
| Average .187 | Average deviation 4.5 |

*Eight analyses, or 32 per cent, fall without the average deviation.

TABLE III. FRACTIONATION OF THE IODINE CONTENT OF 3 ML. OF HUMAN SERUM

| NON PROTEIN-BOUND IODINE | PER CENT DEVIATION | PROTEIN-BOUND IODINE | PER CENT DEVIATION |
|---|---|---|---|
| Patient 1 | | | |
| .09 | - 5.3 | .25 | - 3.8 |
| .11 | +15.8 | .25 | - 3.8 |
| .10 | + 5.3 | .30 | +15.4 |
| .08 | -15.8 | .24 | - 7.7 |
| Average .095 | Average deviation 10.5 | Average .26 | Average deviation 7.7 |
| Patient 2 | | | |
| .11 | - 2.9 | .23 | - 1.7 |
| .11 | - 2.9 | .24 | + 2.6 |
| .11 | - 2.9 | .23 | - 1.7 |
| .12 | + 4.3 | .24 | + 2.6 |
| .12 | + 4.3 | .23 | - 1.7 |
| Average .114 | Average deviation 3.5 | Average .234 | Average deviation 2.1 |
| Patient 3 | | | |
| .18 | +10.3 | .27 | + 1.5 |
| .15 | - 8.0 | .26 | - 2.3 |
| .17 | + 4.3 | .27 | + 1.5 |
| .18 | +10.3 | .28 | + 5.3 |
| .15 | - 8.0 | .28 | + 5.3 |
| .15 | - 8.0 | .24 | - 9.8 |
| Average .163 | Average deviation 8.1 | Average .266 | Average deviation 4.3 |
| Total average deviation 7.2 | | Total average deviation 4.4 | |

nonprotein-bound blood iodine we find that the normal range extends from 1 to 3 micrograms per cent.

When patients have been receiving appreciable quantities of iodine in any form the protein-bound fraction is increased, roughly in proportion to the amount of iodine they have been taking and the length of time they have been taking it. This renders an interpretation of the values so obtained more difficult. Therefore it is desirable for the clinician to draw blood for an iodine analysis before the patient has been placed on any form of iodine medication.

## SUMMARY

A method for the determination of the blood iodine has been presented which answers the requirements of the clinical laboratory.

We wish to express our appreciation to June M. Swanson, B.S., and Isabelle A. Bucklew for their valuable technical assistance.

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# BRONCHIECTASIS: ITS CURATIVE TREATMENT BY PULMONARY RESECTION

## An Analysis of Ninety-six Cases

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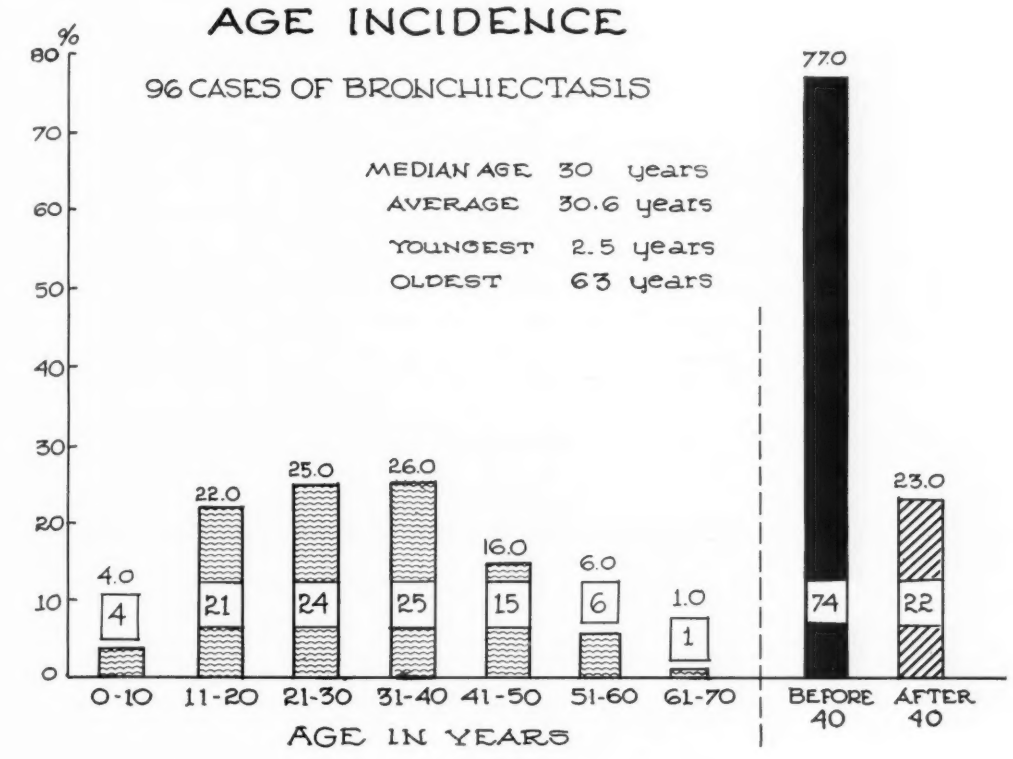
BRONCHIECTASIS is an important chronic pulmonary disease, which vies with tuberculosis as the most common.[8, 11] It may affect persons of any age but characteristically begins in childhood or early adult life after an attack of atypical pneumonia. Symptoms are chronic, progressive, disagreeable, and often disabling. Conservative treatment is palliative but never curative in the established case. Recent advances in thoracic surgery have resulted in the development of a technique of partial or total pulmonary resection which makes possible eradication of the disease in many cases. An analysis of our experience in 96 cases in which 105 pulmonary resections were performed from January, 1933, to May, 1948, indicates that this treatment is not hazardous and gives satisfactory results in a high proportion of selected cases.

Bronchiectasis, as the name implies, is characterized by dilatation of a portion of the bronchial tree. Whereas in some, and probably in many, early cases the bronchial infection causes only functional and reversible[3, 12] dilatation in the bronchi because of relaxation of the bronchial musculature and elastic tissue, later the dilatation becomes irreversible as the result of destruction of these contractile components and their replacement by fibrous tissue. The bronchi then become rigid, noncontractile tubes. Associated with dilatation and recurrent inflammation of the bronchi are inflammation, atelectasis, and destruction of adjacent and peripheral pulmonary tissue.

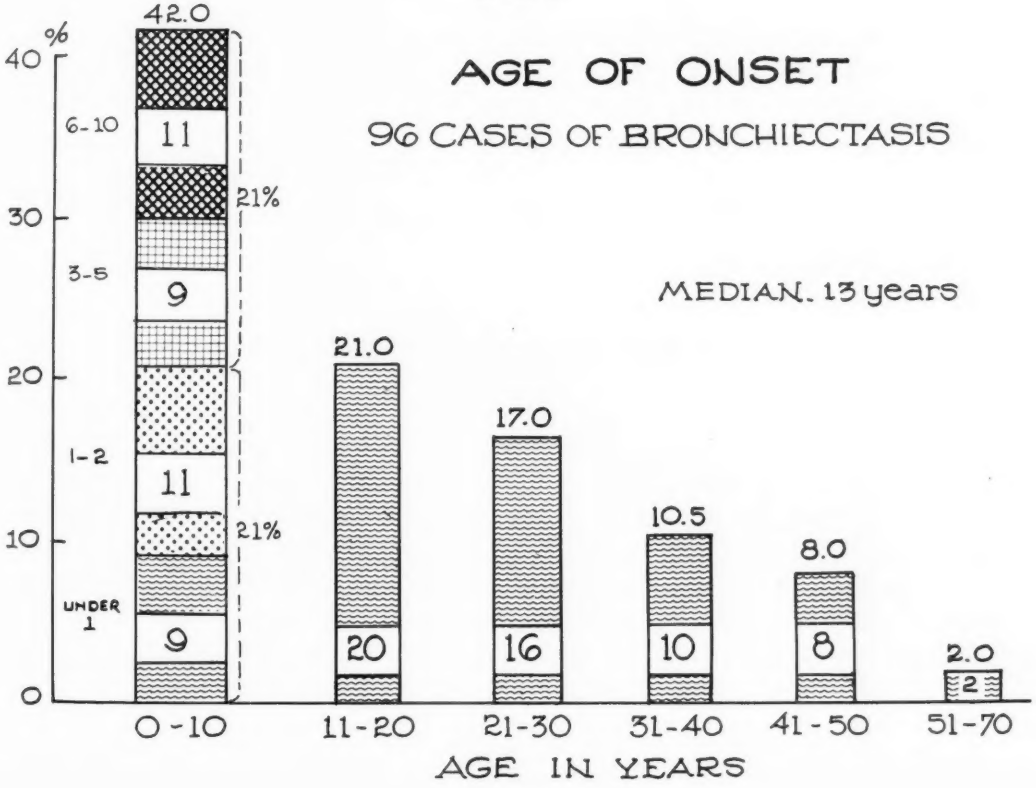
Bronchiectasis appears to affect both sexes with almost equal frequency. The sex incidence in our 96 cases, as in other reported series, was not remarkable. Fifty-three per cent of our cases were females and 47 per cent were males.

Bronchiectasis is chiefly a disease of the young. As shown in Graph 1, the median and average age of the patients in our series when they consulted us was 30 years. Four were in the first decade, 21 in the second, 24 in the third, 25 in the fourth, 15 in the fifth, and 7 past the fifth decade. These figures are not a fair index of the age groups affected by the disease, as Graph 2 indicates that these patients were much younger when the disease began. The median age of onset of symptoms was 13 years. In 42 per cent the symptoms began before the age of 10 years and in 21 per cent definite symptoms appeared in the first two years of life. The disease is not as uncommon in older people as these figures would indicate since pulmonary resection is rarely considered in patients older than 55 years.

Received for publication, Aug. 4, 1948.

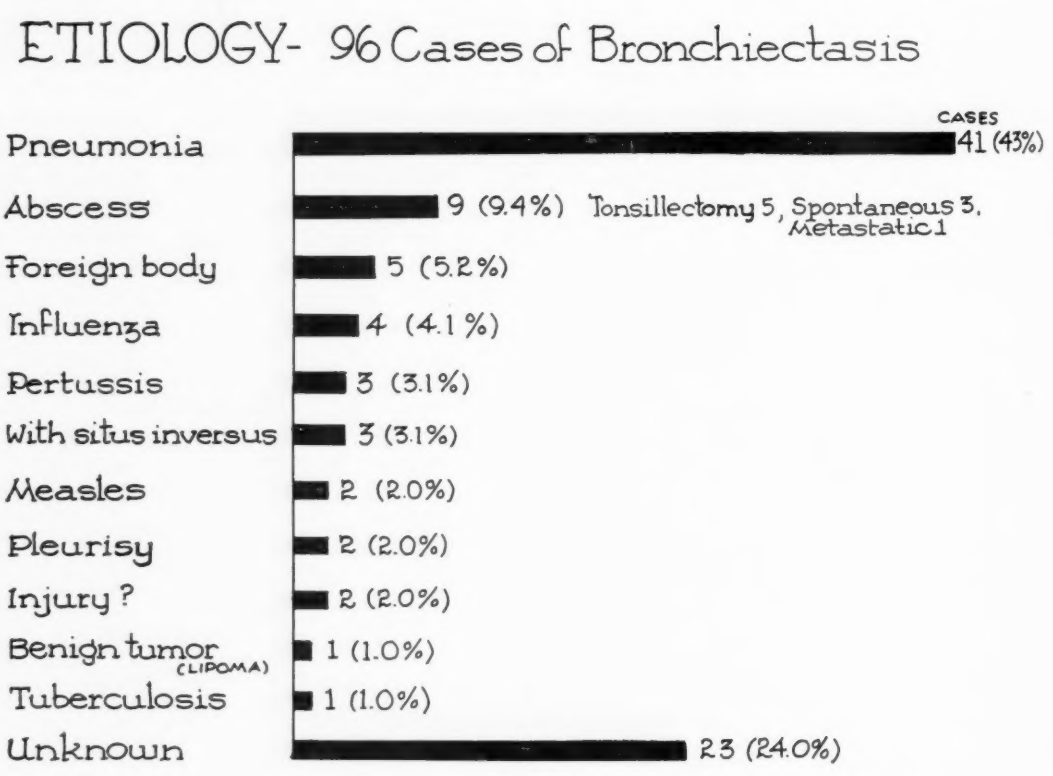



Graph 1.




Graph 2.

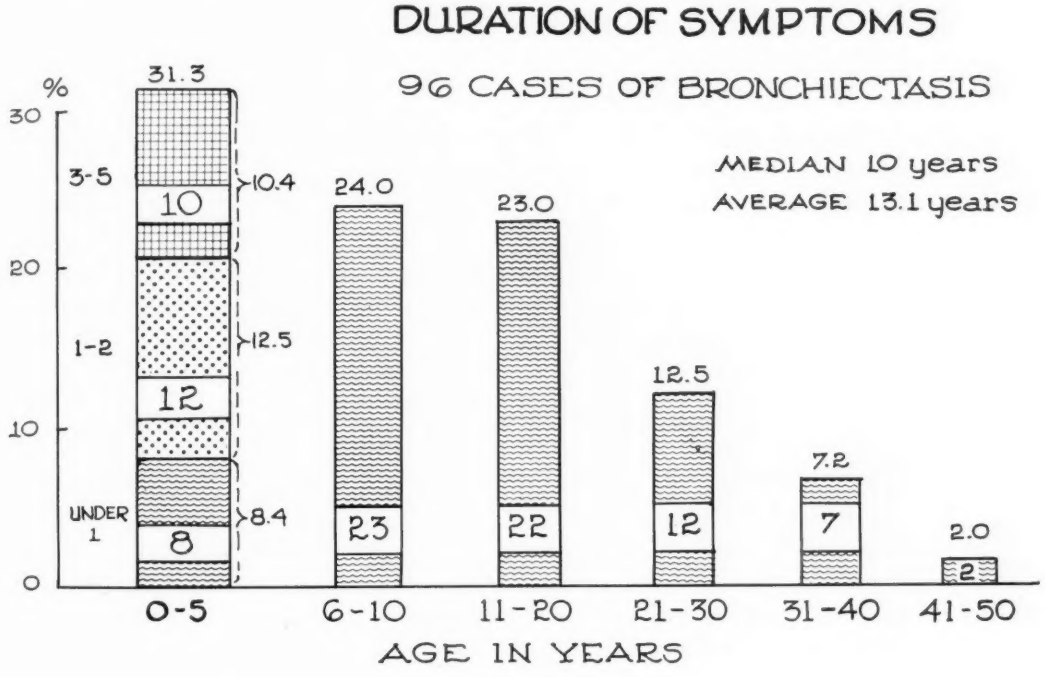
Bronchiectasis usually appears in childhood following an acute lower respiratory infection, most commonly an attack of atypical pneumonia, particularly in infancy when the bronchi are small, the expulsive efforts are weak, and atelectasis is prone to occur.[2] As the atelectasis persists, the retained infected secretions cause inflammation and destruction of the bronchial walls with subsequent dilatation. It is likely that spasm at the root of the bronchial segment contributes to the pathogenesis in many cases.[9] Chronic sinusitis frequently accompanies the bronchial disease but there was no indication in our series that it was an etiologic agent of any importance. As illustrated in Graph 3, there were 41 patients who dated their symptoms from an attack of pneu-




Graph 3.

monia, frequently as a complication of influenza, pertussis, or measles. In 21 cases symptoms began insidiously. In 9 cases the ectasia followed a pulmonary abscess, which was metastatic in 1, spontaneous in 3, and followed tonsillectomy in 5. Four patients attributed the onset directly to influenza, 3 to pertussis and 2 to measles. In 5 symptoms followed aspiration of a foreign body, with the inflammation, atelectasis, and subsequent bronchiectasis presumably due to the bronchial obstruction. A bronchial lipoma was responsible in 1. Symptoms were said by 2 patients to have followed an injury. Hay fever was held responsible by 1 patient. Tuberculosis supposedly initiated symptoms in 1 and pleurisy in 2.

Although in the majority of cases symptoms appear in early life, we believe that the incidence of true congenital bronchiectasis is low. In our series there were 3 patients with associated situs inversus and chronic sinusitis (Kartagener's triad)[1] in all of whom symptoms began early, suggesting a congenital basis in this small group. A somewhat more common related lesion, in our experience, is congenital cystic disease which manifests itself as either single or multiple large cysts, or there may be a severe cystic type of bronchiectasis appearing early in life and usually involving an entire lung which likewise suggests a congenital origin.




Graph 4.

As suggested previously, the duration of symptoms is often prolonged. In our series (Graph 4) the average duration of symptoms for all patients was 13 years and in 9 cases symptoms had existed over 30 years. Cough and expectoration may persist from the outset, though in many cases symptoms entirely disappear for a time and become prominent only after repeated attacks of bronchitis or bronchopneumonia. Even at this stage there may be long intervals of remission when symptoms are minimal. However, the number and severity of colds are likely to increase each winter and the residual symptoms of cough, expectoration, and hemoptysis become more prominent. In many of these young persons the disease progresses unrecognized and is diagnosed as "chronic bronchitis." This diagnosis should never be made until a bronchographic mapping has ruled out the presence of bronchiectasis.

The chief complaint of patients with bronchiectasis is usually chronic cough with expectoration. Expectoration characteristically varies with changes in position as drainage is facilitated. The quantity of sputum may be as much as

12 ounces or more daily. Hemoptysis is frequent and sometimes massive. Foul sputum usually occurs only in the most advanced cases. A sweet taste may be noted. In that smaller group with "dry bronchiectasis," sputum is minimal and the chief clinical manifestation may be hemoptysis or paroxysms of violent coughing. Symptoms of chronic sinusitis may be prominent.

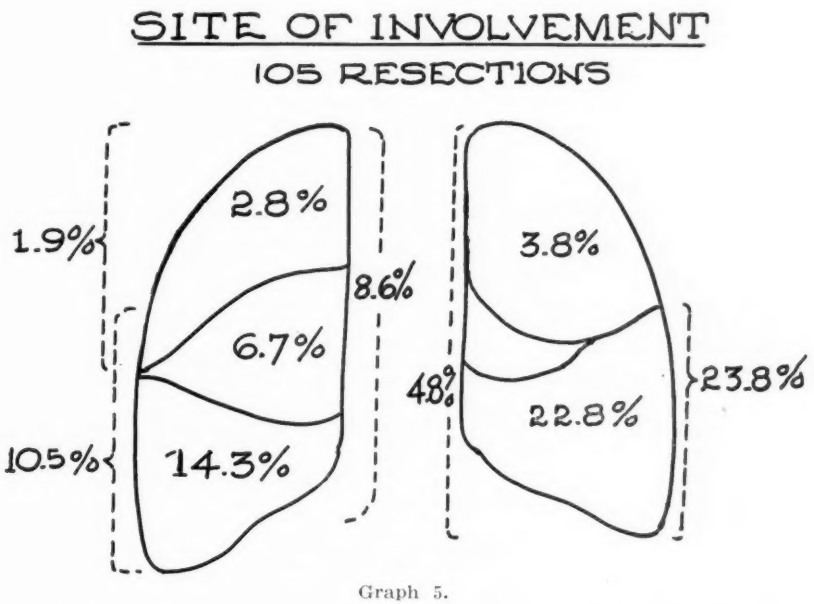
The physical findings and plain roentgenograms are often disarmingly undemonstrative. Clubbed fingers occur only in the advanced cases. Signs of sepsis are rarely prominent. Pulmonary signs are usually limited to moderate changes in the breath sounds, with fine or coarse râles in the area of involvement. In the plain roentgenogram the characteristic sign is increased bronchovascular markings in the involved area. The sedimentation rate may be elevated, but a significant leucocytosis is uncommon. Sputum examination is significant chiefly because specific organisms are not present.

The diagnosis of bronchiectasis is made on the basis of the symptomatology and the physical, roentgenographic, bronchoscopic, and bronchographic observations. The apparent paradox of severe symptoms and minimal physical findings and roentgenographic abnormalities should suggest the diagnosis of bronchiectasis because other lesions manifesting similar symptoms such as tuberculosis, pulmonary abscess, or bronchiogenic carcinoma usually produce definite physical or roentgenographic alterations. Occasionally a shadow suggesting pneumonitis, emphysema, atelectasis, or visible cystic areas will increase the suspicion of bronchiectasis. Bronchoscopy may demonstrate organic obstruction, changes in the bronchial mucosa, and the presence of purulent secretions. As a diagnostic procedure it is only confirmatory.

The essential diagnostic method is bronchography, which is obtained by fluoroscopy and roentgenography following the instillation of a radiopaque substance (lipiodol) into the bronchial tree. It is essential that the entire bronchial tree be satisfactorily filled. The full extent of the pathologic process must be outlined and all normal lung sharply delineated. Only with this information can one proceed confidently with radical surgical procedures. As far as possible, normal lung must be preserved. Diseased segments unrecognized and not removed will cause persistence of symptoms and sometimes serious complications. For satisfactory mapping the oil must be instilled into the lower portion of the pharynx or trachea through a catheter while the patient's position is changed as needed under the fluoroscope. Fluoroscopy frequently reveals important additional information concerning functional changes in the bronchial tree.[9] Both lungs may be filled at the same time. A posteroanterior and two oblique views will satisfactorily outline the entire bronchial tree. The importance of complete and accurate bronchography cannot be overemphasized. When properly done the hazards are minimal. Every patient with symptoms of chronic cough and expectoration, with or without hemoptysis, should have a bronchographic mapping unless some other definite diagnosis is made by other means.

The type of treatment employed is based on the severity of the symptoms, the degree and extent of ectasia, and the age and physical condition of the patient. Medical treatment will not be discussed here. Significant palliation

may be obtained by conservative measures but a cure of the established case is never effected. There are numerous reports in the literature indicating that the morbidity and mortality rates in medically treated patients are high.[4, 13, 14] Minimal symptoms, however, do not warrant a radical attack. In the presence of significant symptoms surgical therapy must be considered. Pulmonary resection is the only form of surgical therapy which has proved satisfactory. The more localized the disease, the more amenable it is to resection. The more diffuse the pulmonary involvement, the more difficult the surgical problem but careful segmental and lobar resections can eradicate even bilateral disease involving several lobes. In bilateral bronchiectasis if the major involvement is on one side, the degree of relief obtained following resection of this portion of the pulmonary bed may be sufficient to obviate the necessity of resection on the second side. If extensive bilateral resections are contemplated, it is imperative that the residual lung be free of bronchiectasis. Resection is generally limited to persons less than 55 years of age because the results in older patients are not as satisfactory and it is possible to obtain symptomatic relief in many elderly patients by conservative measures.




Graph 5.

In Table I are listed the portions of the lung removed in the 105 resections performed on the 96 patients in this series. These data are graphically presented in Graph 5. In 4 instances bilateral operations were performed. In some additional cases operation on the contralateral side is indicated. In 7 instances minor segments of adjacent lobes were resected in addition to the major resections listed in the table. There were 14 primary pneumonectomies; in 6 additional cases the remaining pulmonary tissue on one side was removed secondary to primary lobectomy on the same side, thus effecting total pneumonectomy in these additional cases. The operative techniques are illustrated in

TABLE I. SITE OF RESECTION IN ONE HUNDRED FIVE CASES OF BRONCHIECTASIS

| PORTION RESECTED | NUMBER OF CASES |
|---|---|
| Left lower lobe | 24 |
| Left lower lobe and lingula | 23* |
| Left upper lobe | 4 |
| Right lower lobe | 15 |
| Right lower lobe and right middle lobe | 11 |
| Right middle lobe | 7 |
| Right upper lobe | 3 |
| Right upper lobe and right middle lobe | 2 |
| Left lung | 5 |
| Right lung | 9 |
| Lingula and basal portions of left lower lobe | 2 |
| Total | 105 |

*In three patients with situs inversus these structures were present in the right side of the chest.

Graph 6. Segmental resection of the lingula was performed on numerous occasions. Resection of the basal divisions of a lower lobe, retaining the dorsal division, was done in only 2 cases with segmental atelectasis occurring in 1 case postoperatively. This procedure seems to have a limited field of usefulness as complications are frequent.

Pulmonary resection is admittedly a formidable surgical procedure but the improvement in results over the years has been striking.[5, 6, 10] Increasing experience in the evaluation and management of these patients, technical surgical refinements, effective antibiotics, and the increasing use of whole blood have contributed materially to the improvement in results.

The year 1942 marked a turning point in our surgical experience with this disease. Tourniquet ligations and cautery excisions were completely abandoned for hilar dissections and segmental resections. In addition, effective antibacterial agents, first the sulfonamides and then the antibiotics, were regularly used. Pneumonia and hospital deaths dramatically disappeared (Table II and Graph 7). In the 13 resections performed prior to 1942, pneumonia occurred 5 times and there were 6 hospital fatalities. In 92 resections performed since that time pneumonia has occurred twice and there has been no hospital death

TABLE II. ANALYSIS OF POSTOPERATIVE COMPLICATIONS BY PERIODS

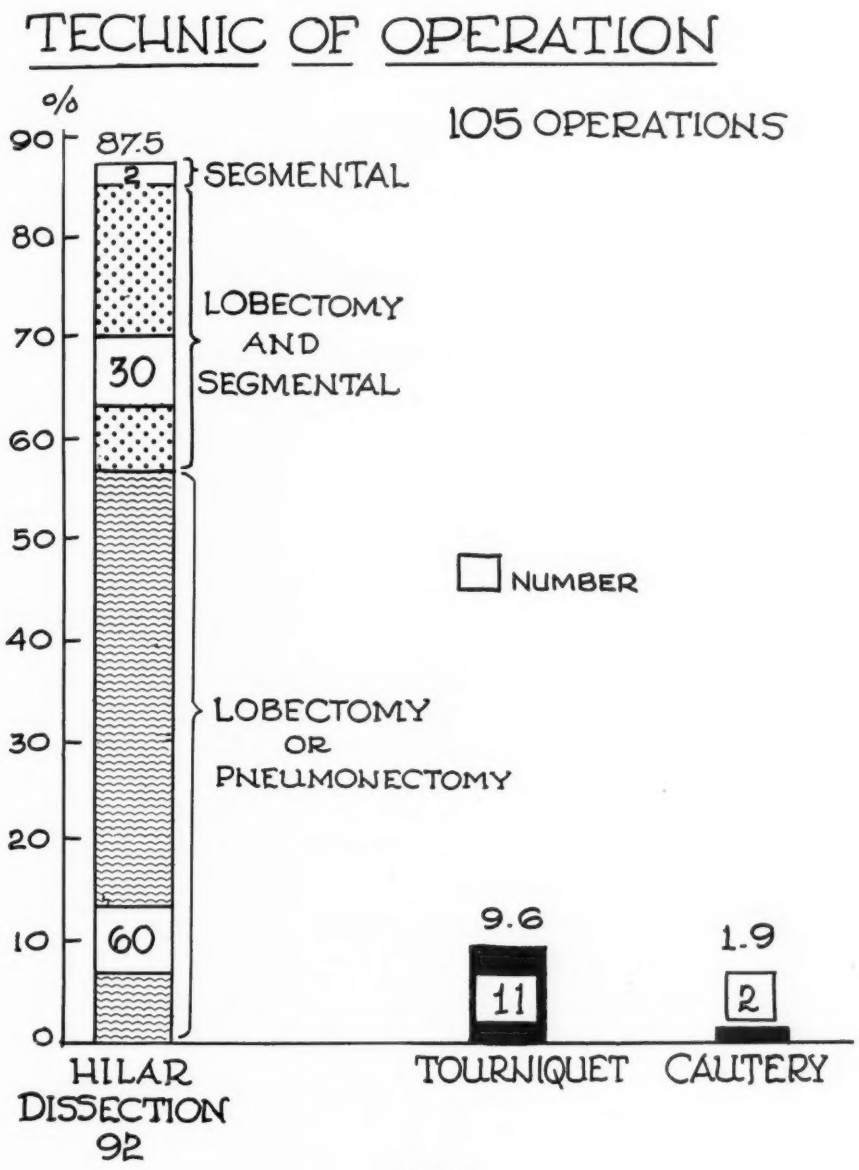
| YEARS | CASES | EMPYEMA (PER CENT) | FISTULA (PER CENT) | PNEUMONIA (PER CENT) |
|---|---|---|---|---|
| 1933 to 1941 | 13 | 55.5 | 40.0 | 38.5 |
| 1942 to 1946 | 59 | 30.5 | 15.3 | 2.4 |
| 1947 to 1948 | 33 | 15.1 | 6.0 | |

TABLE III. ANALYSIS OF POSTOPERATIVE COURSE BY PERIODS

| YEARS | CASES | DAYS FEVER 99.6° F. AVERAGE | DAYS FEVER 99.6° F. MEDIAN | POSTOPERATIVE HOSPITAL DAYS AVERAGE | POSTOPERATIVE HOSPITAL DAYS MEDIAN |
|---|---|---|---|---|---|
| 1933 to 1941 | 7* | 19.3 | 22 | 27.1 | 25 |
| 1942 to 1946 | 59 | 9.0 | 8 | 17.1 | 13 |
| 1947 to 1948 | 33 | 7.9 | 5 | 14.4 | 10 |

*Hospital deaths omitted.

(Graph 8). There has also been a gratifying reduction in the incidence of postoperative complications. It will be seen by comparing the figures in Table II for the period 1933 to 1941 with those for 1947 and 1948, that the incidence of empyema has been reduced to almost one-fourth of the previous figure, whereas the incidence of bronchial fistula has dropped to one-seventh

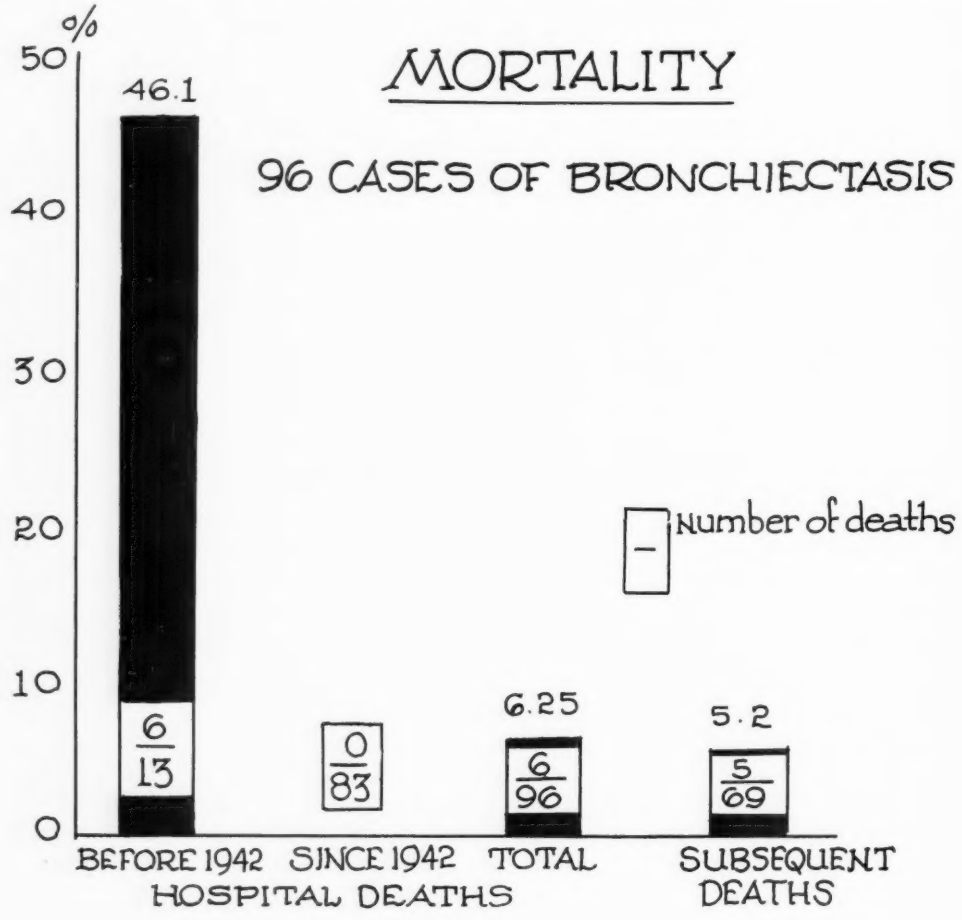



Graph 6.

(Graphs 9 and 10). Table III indicates that the morbidity rate and duration of hospitalization have been strikingly reduced. The average period of hospitalization has been halved and the average period of morbidity reduced to less than half. The median period of postoperative morbidity has dropped

from 22 to 5 days, and the median period of postoperative hospitalization from 25 to 10 days (Graphs 11 and 12).

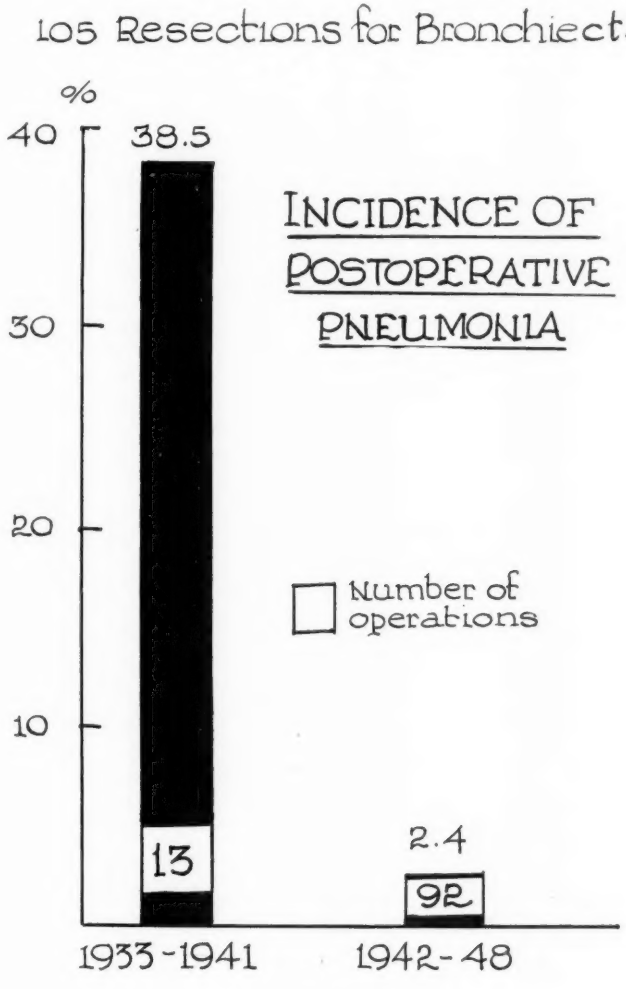
The virtual disappearance of postoperative pneumonia, the chief cause of death, was coincident with the introduction of effective antibacterial agents and the institution of routine hilar dissection. The reduction in the incidence of empyema and fistula has been remarkable, though not as abrupt. Further technical refinements, and the more effective use of antibiotics, have contributed to this improvement. It has become increasingly apparent, however,




Graph 7.

that in the cases of partial pulmonary resection prompt re-expansion of the remaining pulmonary tissue to fill the hemithorax is of the utmost importance in the prevention of these complications. In order to accomplish this the bronchial tree must be constantly cleared of secretions. At operation two intercostal tubes are placed, one anterior to the apex and the other posterior and low in the paravertebral gutter. These tubes are connected to a negative suction and remain for a few days until the lobe is well expanded and the pleural drainage minimal. Complete re-expansion usually occurs within

twenty-four to seventy-two hours. Postoperative pain has been reduced by the injection, at the time of operation, of all the intercostal nerves with a long-acting local anesthetic agent. This facilitates the encouragement of voluntary coughing which is the most important postoperative maneuver. The patient must be closely observed, examined, and assisted in coughing at frequent intervals the first few days. Nothing can replace this constant attention. Whenever spontaneous bronchial clearing is inadequate, intratracheal catheter suction should be used. This may be done simply and repeatedly

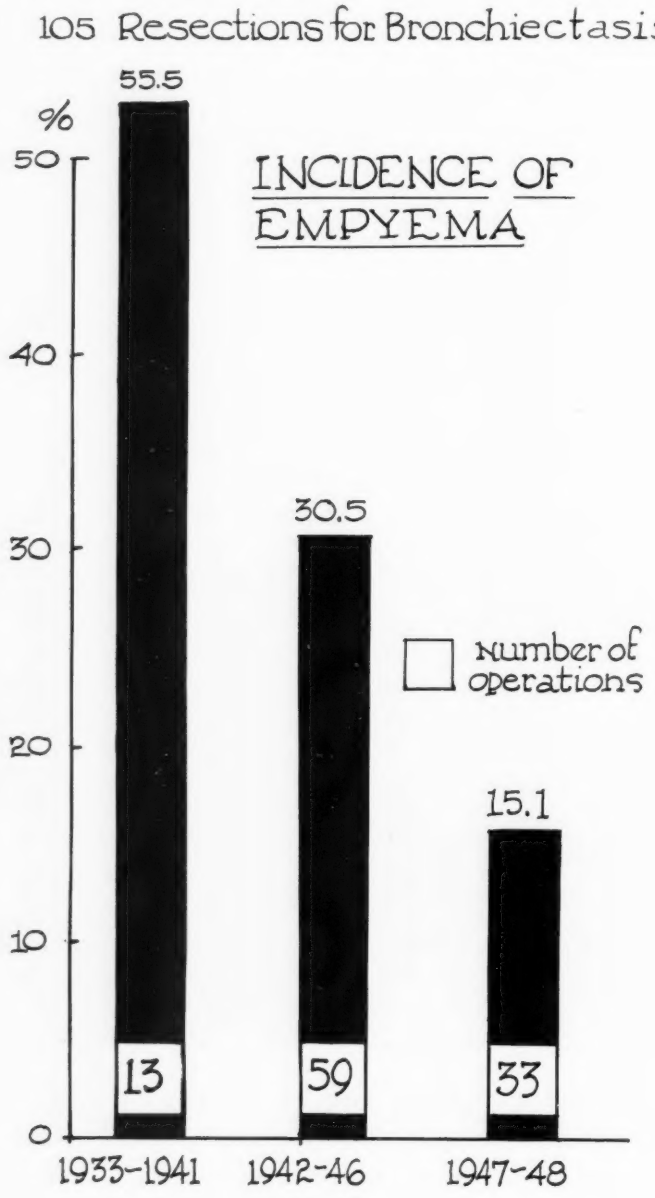



Graph 8.

at the bedside. The clearing is almost always satisfactory and bronchoscopy is seldom necessary although occasionally it is invaluable. Complete prevention of empyema following total pneumonectomy, with the resultant empty hemithorax, will depend chiefly on a more effective antibacterial regimen. This complication occurred in 4 of the 20 cases in which pneumonectomy was done. The liberal use of whole blood preoperatively to correct anemia and to restore diminished blood volume in the debilitated,[7] as well as prompt

operative and postoperative replacement of all blood lost has prevented shock, hastened convalescence, and, we believe, aided materially in aborting infection.

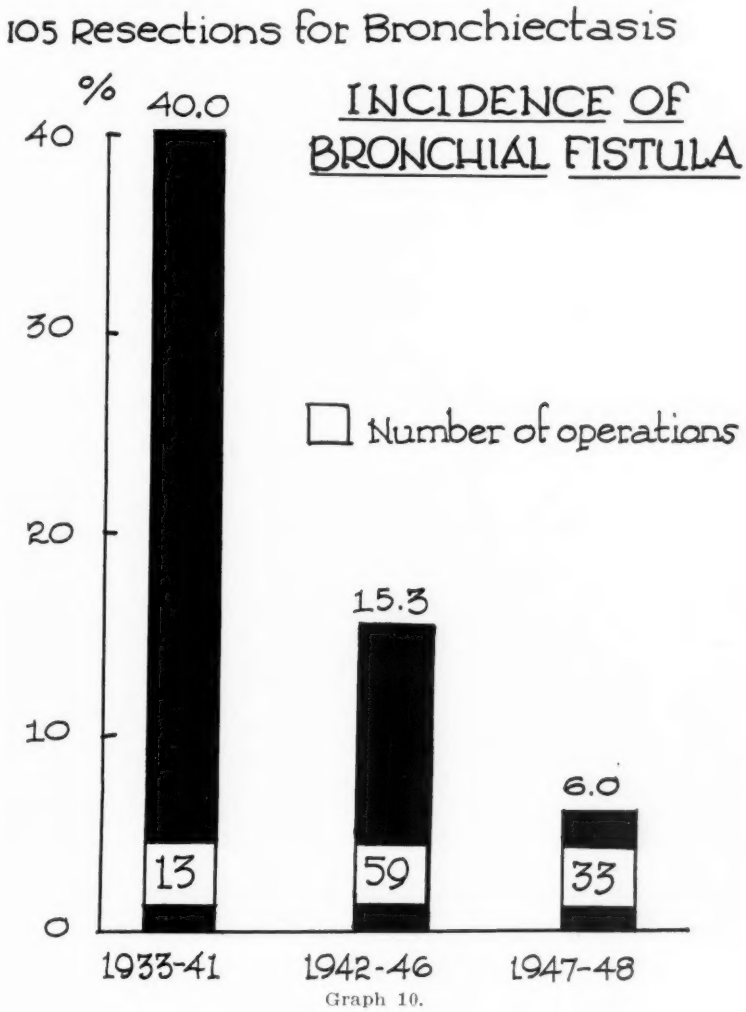
Final results in this group of cases are gratifying on the whole, although some sobering lessons have been learned. Of the 6 hospital deaths occurring early in the series, 1 was an operative death from asphyxia caused by ex-




Graph 9.

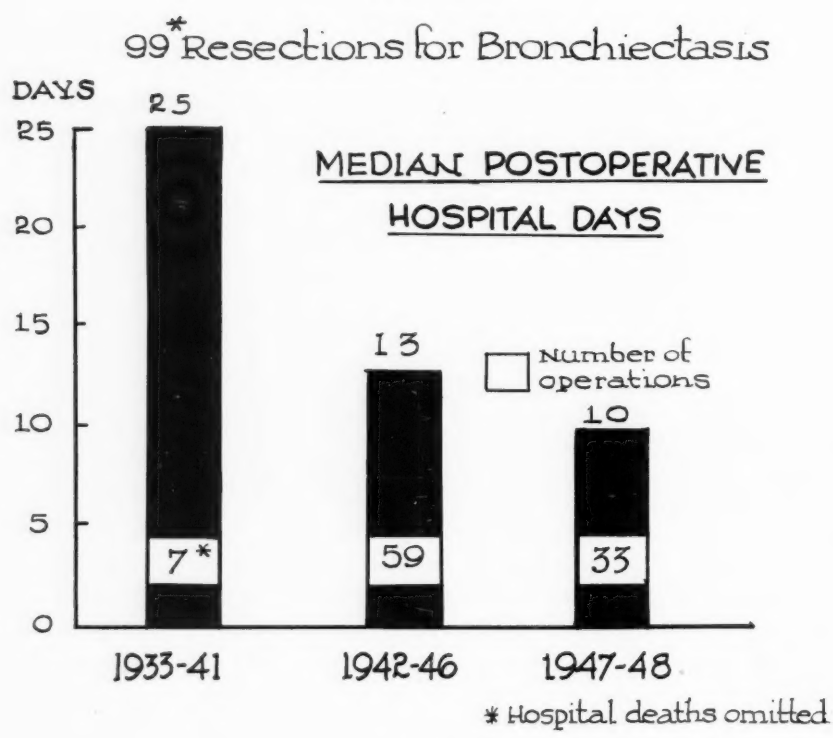
cessive secretions; pneumonia was a major cause in 3; 1 was caused by acute glomerulonephritis and heart failure, and 1 resulted from a transfusion reaction. Five deaths have occurred subsequent to hospital discharge. One patient died of pneumonia after four asymptomatic years. Another died after

the second attack of pneumonia in a year. This patient had extensive bilateral disease for which bilateral operation was performed. He had some bronchiectasis in the remaining upper lobes. This had not been correctly evaluated preoperatively and pulmonary resection was probably not indicated. A third patient, a woman with asthma, died two months postoperatively of acute dyspnea. She had had a pneumonectomy, and ill-advised attempts had been made to aspirate the hemithorax. A fourth died in an accident, and the fifth died during a subsequent operation for an unrelated disease.

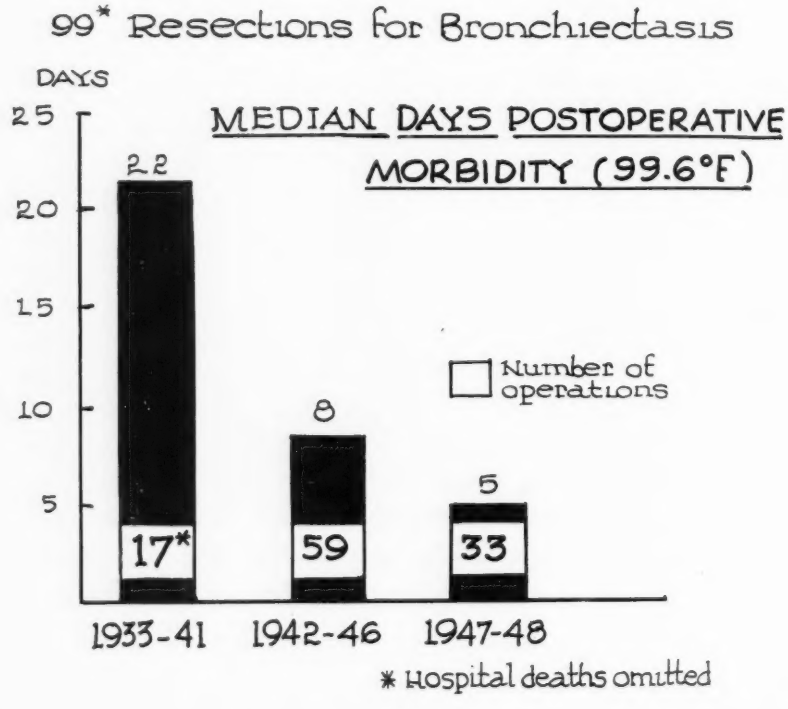



Graph 10.

In 3 cases the follow-up has been inadequate. The remaining 82 patients have been followed from two months to twelve years postoperatively with an average of 2.19 years (Graph 13). Forty-three patients, 44.9 per cent of the entire series, are symptom-free and enjoy no limitation of activity. Twenty-one additional patients, or 21.9 per cent, have minimal symptoms of cough, or mild dyspnea on exertion. Twelve patients, or 12.5 per cent, are



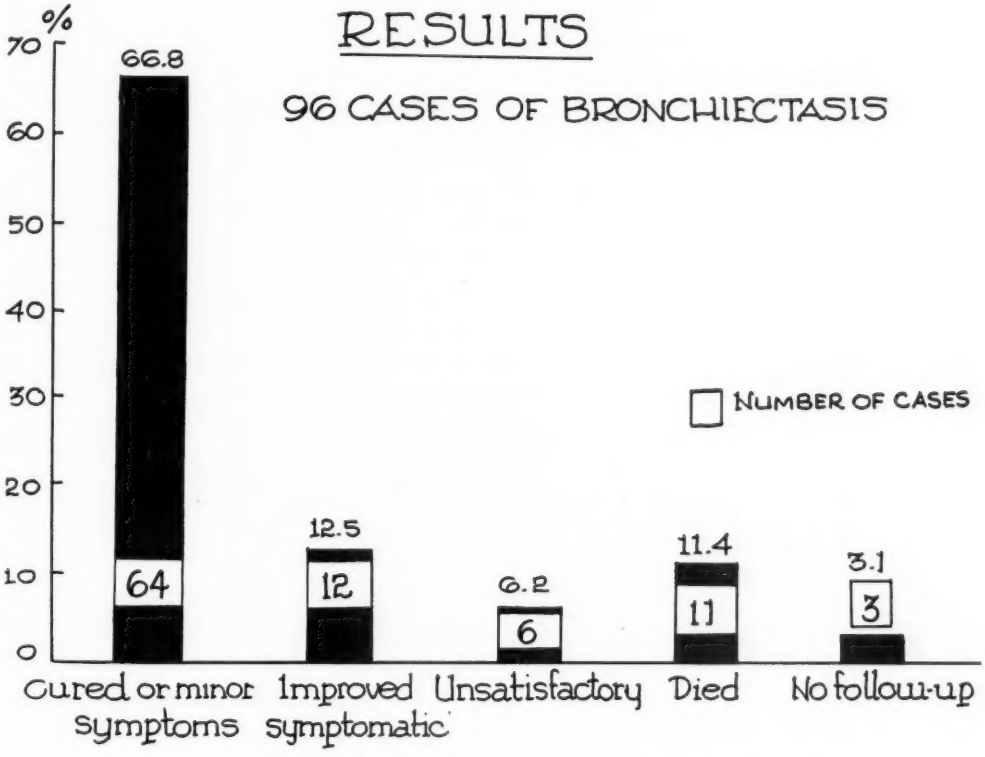

Graph 11.




Graph 12.

definitely improved but have moderately disabling symptoms. In five of these the symptoms are primarily due to contralateral bronchiectasis which could be resected. Thus, improvement of a satisfying degree has been noted in 76, or 79.3 per cent, of the entire group.

Of the 6 remaining patients 2 have postpneumonectomy empyemas which are under treatment. In a patient with hypertension and recent cerebral thrombosis, residuals are present following extension of the thrombosis postoperatively. Persistent fever made operation imperative in this admittedly poor-risk patient. In one case in which bilateral lower lobe resection was performed, residual disease is present in the right middle lobe and the lingula. In the last two patients, both with bilateral disease, surgical attack on the second side has been prevented by atelectasis and bronchiectasis developing postoperatively in the remaining pulmonary tissue on the operated side which necessitated pneumonectomy on that side.




Graph 13.

This final group of 6 cases must be classed as definitely unsatisfactory results, though in the 2 cases of empyema the condition is correctable. These experiences emphasize the utmost importance of complete lung mapping preoperatively, and the necessity for prompt expansion of residual pulmonary tissue postoperatively to prevent atelectasis and subsequent additional bronchiectasis. In 4 patients with persistent postoperative atelectasis, bronchiectasis developed requiring removal of the remaining portion of that lung.

## COMMENT

It is not our intention to minimize the gravity of treatment by pulmonary resection for bronchiectasis. Accurate diagnosis is essential. Preparation must be thorough. The operative technical difficulties may be great. Expert anesthesia and an adequate supply of blood are required. Postoperative management demands a degree of vigilance scarcely equalled in any other thoracic or general surgical condition.

The progressive improvement in results with the reduction in morbidity and the practical elimination of fatalities as a result of the procedure make pulmonary resection an attractive therapeutic method. In our series there has been no hospital fatality in the last 92 operations. Subsequent death from pulmonary disease has occurred in 3.1 per cent of the cases. The incidence of irremedial unsatisfactory results has been 4.2 per cent. With present experience and facilities most of these unsatisfactory results should be completely prevented in the future. It, therefore, seems fair to state that pulmonary resection is indicated in any established case of bronchiectasis provided the disease is neither too mild nor too extensive, the patient is not elderly, or medical conditions are not contraindications.

## SUMMARY

Bronchiectasis is a common, chronic, progressive pulmonary infection characterized by cough, expectoration, and recurrent acute pulmonary infections. The disease usually begins in early life and, once established, though it may be relieved by conservative treatment, it can be cured only by resection of the diseased portions of the lungs. This is a formidable operative procedure but it gives excellent results in a high percentage of properly selected cases. With present methods mortality is minimal and morbidity is low.

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# THE ANATOMIC GUIDE TO THE INTERSEGMENTAL PLANE

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THE ideal in excisional surgery is the removal of all damaged or diseased tissue and the conservation of all functional normal tissue. In pulmonary surgery this ideal is approached in segmental resection for noncancerous disease. Churchill and Belsey[1] first projected the thought of segmental resection and applied the idea to removal of the lingula, a portion of the lingula, and the basal segments of the lower lobe conserving the superior segment, in bronchiectasis. After the segmental bronchus had been occluded, they used clamps across the plane delineated by expansion of the remaining portion of the lobe, and oversewed the line of resection. Planes between other segments often run deep in a lobe and are difficult to clamp. The plane is not sharply adhered to by the clamp method, and either some tissue is left deprived of its blood supply or tissue of another segment is damaged. Suturing of the cut margins limits expansion of the remaining lung and the compensatory filling of the remaining space by adjacent lung tissue.

Overholt and Langer[2] have extended segmental resection to all segments and some subsegments to conserve healthy tissue and in so doing have abandoned the use of clamps, staying accurately in the intersegmental plane. This is done by blunt dissection, using the index finger and the thumb. Complications after segmental resection may be more numerous than after lobectomy if there is leakage of air or passage of infection from exposed alveolar or bronchial tissue when the plane has not been promptly found or has been deviated from.

Surgeons who are doing segmental resections know that there is difficulty at times in finding this plane. There is danger of entering some other intersegmental plane, or of dissecting for a distance in alveolar tissue. After occlusion of the segmental bronchus has been accomplished, inflation of the remaining lung helps to demarcate the intersegmental plane. Inflammatory changes, present in most lungs where resection is indicated, make the intersegmental plane less easy to discover and more difficult to follow without damage to lung parenchyma. The injection of dye into the segmental bronchus of the segment to be removed has been advocated and used by some. Obstruction by tumor or pus might make this impractical, and theoretically the danger of contamination would be increased. At The Overholt Thoracic Clinic we have had no experience with this technique. No method for delineation of the plane known to us has been completely satisfactory. The arrangement of the intersegmental veins is not disturbed by inflammation or other pathology and they continue to be accurate guides under all conditions.

It has been known for some time that when dissection was done in the proper plane, a vein was to be found running between the two adjacent surfaces. I

Presented in the Forum on Fundamental Surgical Problems at a meeting of the Clinical Congress of the American College of Surgeons, Los Angeles, Calif., Oct. 18-22, 1948.

Received for publication, July 19, 1948.

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believed that if some way could be found to commence the intersegmental dissection using the vein as a guide, then there should be no accidental entering of alveolar tissue, removal of segments not involved with disease, nor the leaving behind of devascularized residua. This in turn would mean a marked reduction in the incidence of complications. The concept was first arrived at about April, 1947. Dissections were done on twenty lungs obtained from the pathologic laboratories, and it became apparent that, although there are variations in the number and distribution of the pulmonary veins, there exists a common pattern. There is a definite arrangement whereby a small amount of blood is returned by small veins which closely accompany the centrally situated segmental bronchi and arteries, usually one for each segment, and a large amount of blood is returned by trunks which are found on the periphery of the segment. Where two segments are in apposition these veins are intersegmental in location; elsewhere they are subpleural. It has been noted that when the fissure lines between the contiguous lobes are incomplete, the plane of delineation is often well marked by one of these branches which, although normally subpleural, has been given intersegmental or interlobar position by the fusion of the fissure line. All veins terminate in the larger trunks of the pulmonary veins. Boyden[3] stated several times that the principal distribution of the veins is intersegmental and in discussing the veins of the right lower lobe he pointed out, "Doubtless if the superior segment were separated surgically, ramus V6b could be removed with the superior segment or be left attached to the basal segments."

In view of his extensive report, a detailed description of the dissections at New England Deaconess Hospital in regard to the normal anatomy of the hilar structures is omitted. However, our findings confirm his statements on the venous anatomy. For purposes of utmost accuracy Boyden's descriptions and the numbering system should be adhered to. Operating surgeons will probably dispense with the names and just remember that pulmonary veins are segmental and intersegmental. The former are small and closely accompany the segmental arteries and bronchi, which themselves run side by side. The intersegmental veins do not accompany such structures and can be located in the hilum and followed distally as a practical guide to the intersegmental plane. Thus, a simple technique can be followed in segmental resection of practically all bronchopulmonary segments. While these venous guides are most easily followed in the more commonly performed segmental resections of lingulectomy, removal of the basal segments, or of the superior division of the lower lobe, they prove most valuable in removal of individual basal segments, upper lobe segments, and subsegments. Diagrams are presented to show that the lungs are composed of individual units consisting of a central segmental artery, bronchus, and vein leading into a cone of alveolar tissue. The periphery of the cone features several veins carrying blood centripetally toward the hilum. These are subpleural in position. When such individual segments are combined to form lobes, the surfaces of contact lose their pleural covering and the veins become intersegmental. These intersegmental surfaces become flattened by contact and the lobe and segments undergo alterations in shape varying with

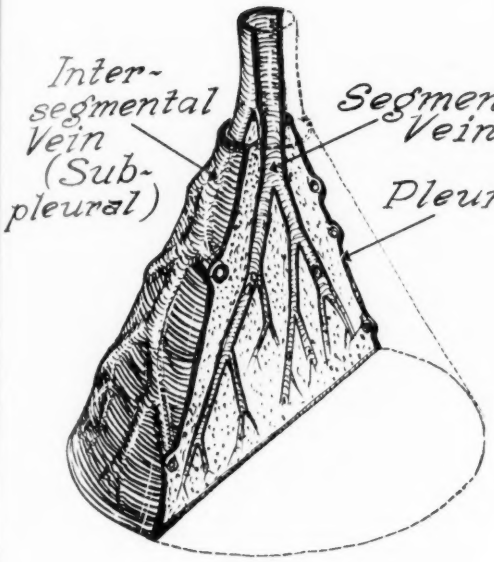


Fig. 1.

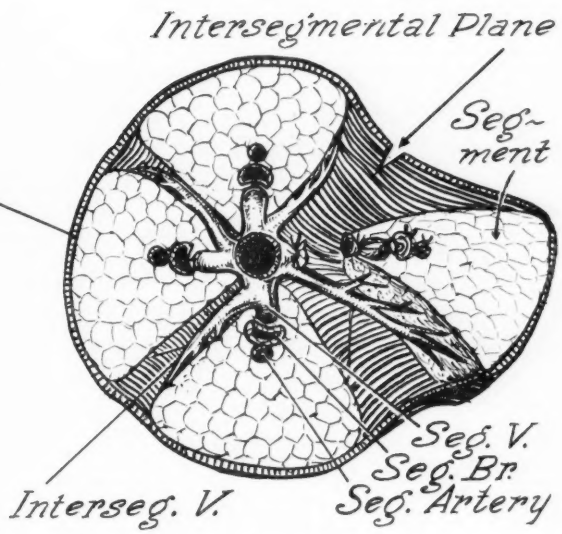


Fig. 2.

Fig. 1.—This diagram represents the bronchopulmonary segment or unit. It consists of a cone of tissue with a central bronchus, artery, and vein, and peripheral collecting veins beneath the pleura. The central bronchus and artery have been omitted for the sake of clarity.

Fig. 2.—This diagram pictures four units combined to form a lobe. Each segment has a central or segmental vein, and the adjacent surfaces of contiguous units are drained by a common intersegmental vein. The segmental bronchus and artery of each unit are shown in cross section.

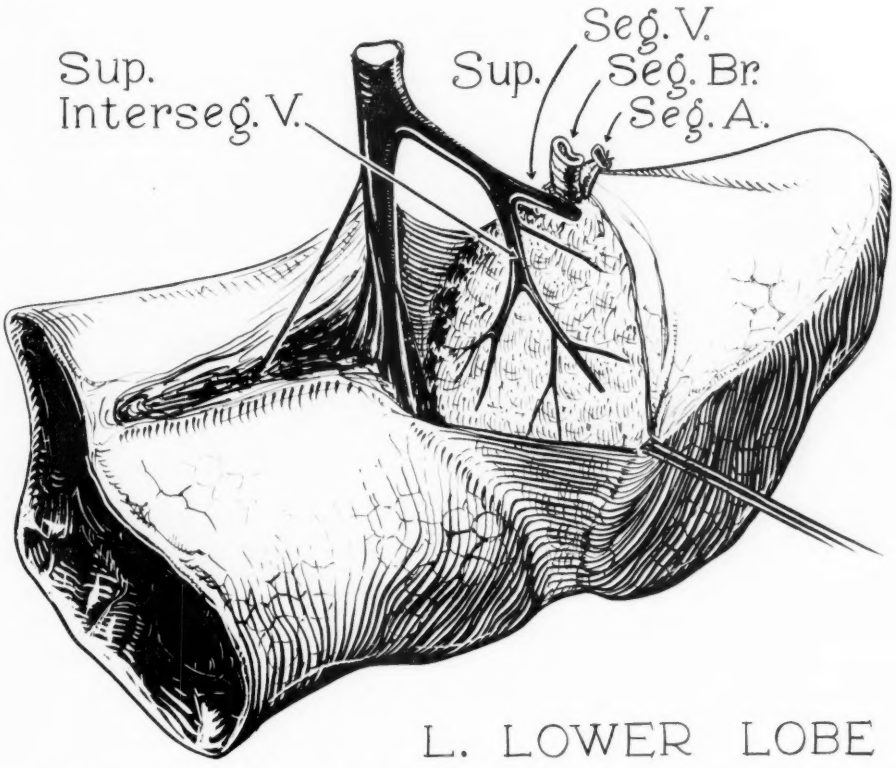


Fig. 3.—This is an anatomic drawing of a dissected specimen illustrating the intersegmental anatomy of a superior segmental resection. The segmental artery and bronchus have been divided. The superior division of the inferior pulmonary vein is shown dividing into a segmental and an intersegmental branch. The intersegmental branch and its tributaries have been followed into the intersegmental plane separating the superior division from the basal segments. Just the pleura remains undivided between the two portions. The vein labeled "Segmental Vein" is the common trunk formed by the branches draining the three subsegments of the superior segment. These branches are both intersegmental and segmental.

their intrathoracic positions. Anatomic drawings of dissected fresh lungs illustrate the intersegmental plane and the venous guide between the basal segments and superior segment of the left lower lobe, and between the lingula and the anterior segment of the left upper lobe.

### TECHNIQUE

Because of inherent variation in the number and size of segments and of the number and course of the veins, it is thought wisest merely to establish the principle of the technique. The segmental bronchus and its artery are found and divided in the usual manner as described by Overholt and Langer.[6] Any

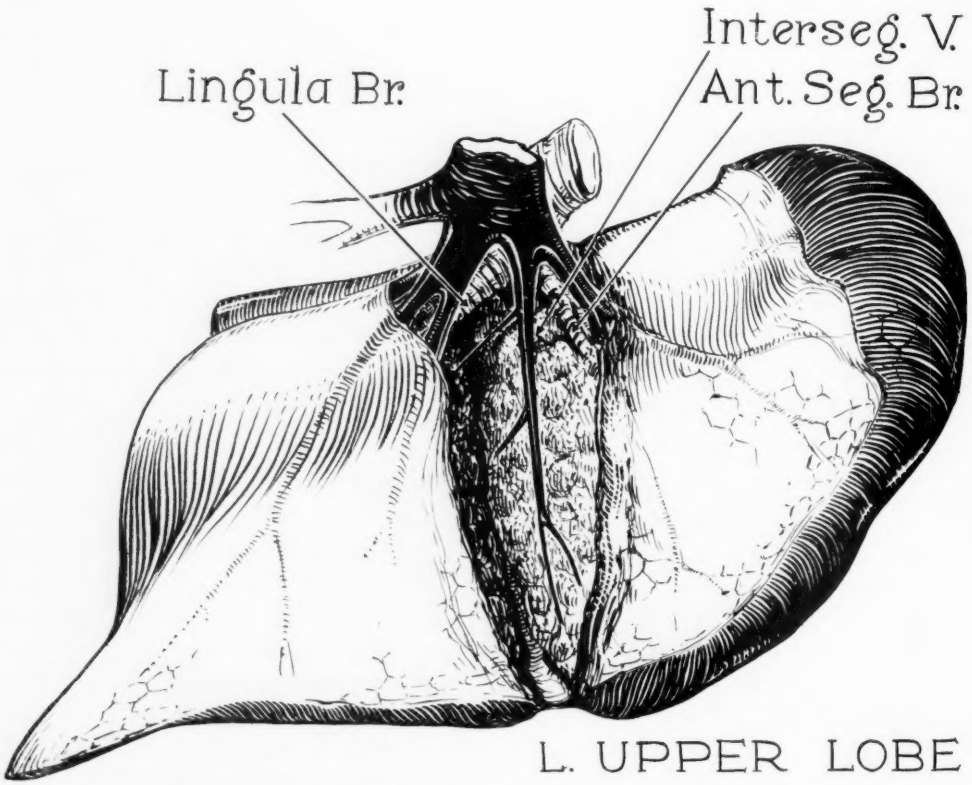


Fig. 4.—This drawing of a dissected specimen demonstrates the intersegmental vein which delineates the plane of dissection between the lingula and the anterior segment. The bronchi of the lingula and of the anterior segment can be seen on either side. Several subpleural veins are illustrated.

subpleural veins and the segmental vein are divided as encountered. The remaining part of the lung is inflated. As the distal divided end of the segmental bronchus is grasped in a clamp, careful observation is made while the apex of the segment is thus retracted. The operator readily finds a vein, or in the case of some segments more than one, which is not accompanying a bronchus and which borders the segment being retracted. He carefully follows this distally, using gentle blunt dissection and dividing the small venous branches coming in as tributaries from the diseased segment. He is dissecting in the areolar tissue

between contiguous segments. Whereas he can dissect on either side of this vein, it is often best to preserve the vein with the healthy tissue. Although the basal segment of a lower lobe is composed of three (left) or four (right) components, each of which is a complete segment in its own right, the group can be treated surgically as one. Thus the common bronchial, arterial, and venous trunks are dissected and divided and the intersegmental vein is sought for in the hilum. In this case, it is the lowermost branch of the superior division of the inferior pulmonary vein as illustrated (see Fig. 3). Because this particular intersegmental vein is so definitely a tributary of the superior segmental vein, it should always be dissected on the basal side, leaving it attached to the superior segment. This new method or variation in the technique of segmental resection has been used by Dr. Overholt with success in several segmental resections, and in determining the plane between upper and middle and upper and lower lobes when the fissure has been incomplete.

## SUMMARY

1. Twenty lung dissections have been done which confirm the previously described intersegmental distribution of most of the pulmonary venous drainage.

2. The veins, for practical surgical purposes, are not given the involved anatomic designations previously used but are simply called segmental and intersegmental.

3. This intersegmental arrangement is utilized in a modification of segmental resection[2, 6] wherein the intersegmental veins of bordering segments are followed distally from the hilum to the pleural surfaces as accurate guides to the areolar planes which separate such segments.

4. Diagrams and anatomic drawings are used to demonstrate pictorially the intersegmental anatomy.

## CONCLUSIONS

By following the intersegmental veins peripherally from the hilum, the operator can enter the intersegmental planes accurately. By so doing, lung tissue is not injured, leakage of air from alveolar damage is almost eliminated, and infection from the exposed parenchyma is greatly diminished.

The incidence of complications such as inexpansible lung, empyema, and bronchopleural fistula should be not higher than that of an uncomplicated lobectomy.

*Note.*—Since this paper was submitted for publication, this method has been used with progressive enthusiasm by Dr. Richard H. Overholt, Dr. Francis M. Woods, and Dr. Norman J. Wilson. It has become the procedure of choice. There has been a definite decrease in the incidence of postoperative complications, and the re-expansion of the remaining lung tissue has been much less of a problem. Complete re-expansion with removal of the intrapleural catheters has occurred in an average of forty-eight hours.

The intersegmental veins can be readily located at the hilar area of segments, one-half segments, and even one-quarter segments and then followed as practical guides to the plane of separation. It is possible that the smallest branches of the veins demarcate the planes between pulmonary units of ever-decreasing size, each of which has the characteristic structure of a pulmonary unit (Fig. 1). It is unlikely that the application of the principle of segmental resection to units smaller than one-quarter segments will be practical.

The writer wishes to acknowledge the encouragement and advice of Dr. Richard H. Overholt.

The anatomic material was obtained from the pathology Laboratories of the New England Deaconess Hospital through the courtesy of Dr. Shields Warren, Pathologist, and Dr. William Meissner, Associate Pathologist.

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# THE USE OF GLYCINE IN THE TREATMENT OF PERIPHERAL VASCULAR DISEASE

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FOLLOWING the ingestion of food, there is, for a variable period of time, an increase in energy output by the body. This phenomenon has been termed "specific dynamic action" by Rubner.[1] The magnitude of the specific dynamic action varies with the substance ingested, with the environmental temperature, and possibly with the nutritional status of the individual. The largest specific dynamic action is that evoked following the ingestion of protein. This rise in metabolic rate averages 20 per cent above the basal level between the third and fifth hours following ingestion and a considerable elevation is still present after seven hours.[2] Rubner contributed some of the most important fundamental work on this subject, and in more recent years, Lusk,[3] Wilhelmj,[4] Borsook,[1] and others have carried out exhaustive studies on the metabolism of protein, regarding its specific dynamic action.

The source of the extra heat production apparently attends and follows the deamination of the protein or amino acid. There has been some difference of opinion as to whether this occurs during deamination or in the metabolism of the deaminized residue. Borsook[1] believed that the specific dynamic action is a composite of two factors, one nearly constant, representing the increased energy production attending the metabolism and excretion of the nitrogen; the other, more variable, and at times the larger fraction, arising from the metabolism of the carbon. This would account for the variations in the specific dynamic action of differennt amino acids; glycine, alanine, phenylalanine, and tyrosine have high values, while valine and histidine are reported to have no effect.[4] The heat produced by these reactions is not utilizable by the organism for muscular work, etc. and, hence, appears only as heat.[3] The site of the increased heat production is in the viscera, probably chiefly in the liver.[14] Since the extremities represent the most important sites for the dissipation of heat, it is reasonable to assume that there will be an increase in peripheral blood flow to the extremities[5, 6, 7] (Fig. 1).

Under constant environmental conditions, there is a simple linear relationship between peripheral blood flow and metabolic rate.[6, 8, 9] Abramson and Fierst[10] studied the effects of food on peripheral blood flow and noted an increase following a protein meal but none subsequent to ingestion of carbohydrate. Gubner, DiPalma, and Moore[11, 12] have recently published their findings on the

Aided in part by the John Harper Seeley Foundation and the Cardio-Vascular Unit, University of Michigan Medical School.

Presented in part at the Forum on Fundamental Surgical Problems, American College of Surgeons, New York, N. Y., September, 1947.

Received for publication, July 7, 1948.

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utilization of specific dynamic action to increase heat production in the body and thereby increase the blood flow through the peripheral vessels. The effects of the ingestion of glycine on skin temperature, the amplitude of peripheral pulsations, and the peripheral blood flow as measured by venous occlusion plethysmography were presented. They concluded that "the ingestion of glycine provided a simple physiologic means to accomplish an effective and sustained increase in peripheral blood flow."[12]

The clinical application of these findings to patients with peripheral vascular disease is obvious. The present investigation was undertaken in an effort to evaluate the use of glycine as an adjunct to the conservative therapy of peripheral vascular disease. Both clinical and laboratory studies were utilized for this investigation.

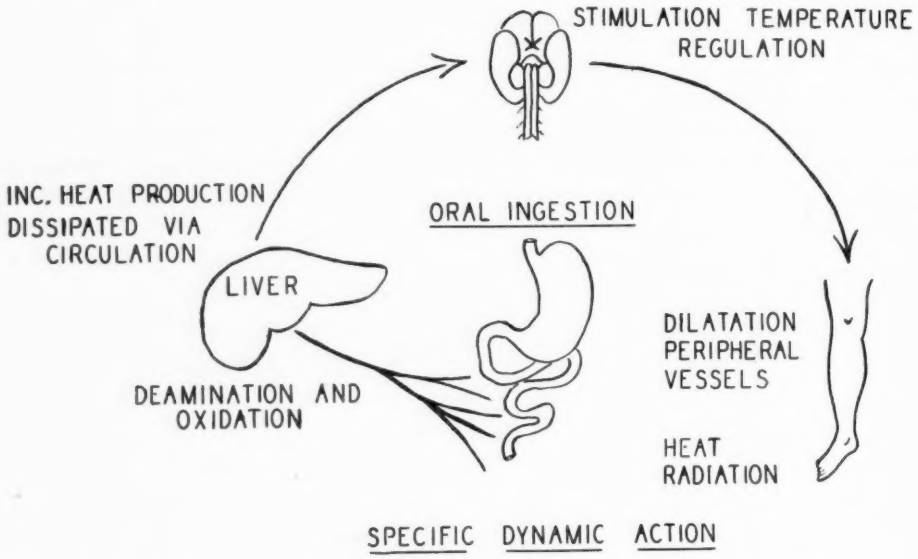


Fig. 1.—Theoretical explanation of how specific dynamic action produces increase in peripheral blood flow.

## LABORATORY STUDIES

Observations of the peripheral blood flow, oxygen consumption, and skin temperature before and after the administration of glycine were made on ten patients with peripheral vascular disease. All studies were conducted in an air-conditioned room, the temperature of which was maintained between 70 and 73° F. To insure a basal state, the patients reported to the laboratory without having taken food or fluids and without having smoked since the previous evening. A minimum of thirty minutes of rest in the recumbent position was allowed prior to testing.

The peripheral blood flow was measured by venous occlusion plethysmography of the affected part. Observations were made in the recumbent position; the water bath of the plethysmograph was maintained at 32° C. by an automatic heating and agitating system. The venous occlusion pressure was determined by trial runs on each patient; usually 10 mg. of Hg below the patient's

diastolic blood pressure was most satisfactory. All recordings were made in ink on a high speed kymograph. Basal blood flow readings were made for a period of twenty to thirty minutes prior to the administration of glycine; a minimum of fifteen determinations was made in the control period. Following the administration of glycine, five to seven determinations were made at fifteen-minute intervals for three hours. Pulse and blood pressure recordings were made at each interval. It was not feasible to carry the study beyond three hours because of the discomfort of confinement in the plethysmograph. This obstacle was also experienced by Abramson[10] and by Gubner and co-workers[11, 12] in their initial work. This was an unfortunate physical limitation, for the effect of protein ingestion apparently continues for a considerably longer period.[2]

Skin temperature recordings were made during the control period and at each fifteen-minute interval following the administration of glycine. The temperatures were taken on both upper and lower extremities. In most instances, a Leeds-Northrup potentiometer was utilized; on several occasions a multiple lead automatic temperature recording apparatus was used but this proved to be somewhat unsatisfactory because the additional confinement led to losing contact at some points as the patient adjusted his position on the table.

Oxygen consumption was measured on a Benedict-Roth basal metabolism machine. Determinations were made in the basal state and at hourly intervals following the administration of glycine.

Twenty grams of glycine (amino-acetic acid) dissolved in 200 c.c. of water were administered in each of the ten studies. Four of the patients had arteriosclerotic peripheral vascular disease, three had thromboangiitis obliterans, two had Raynaud's disease, and one had Raynaud's phenomenon secondary to a possible ruptured cervical intervertebral disc.

*Oxygen Consumption.*—All ten patients demonstrated an increase in oxygen consumption. The greatest increase was at the end of the first hour with an average metabolic rate of 25.7 per cent. The average rate for the second hour

TABLE I

| | BLOOD FLOW (C.C. PER 100 C.C. LIMB PER MIN.) | | | TEMPERATURE CHANGE (°C.) | AVERAGE INCREASE IN METABOLIC RATE |
|---|---|---|---|---|---|
| | BASAL | MAXIMAL | CHANGE | (TOE OR FINGER) | (PER CENT) |
| R. L., Artscl. (foot) | 1.2 | 2.2 | +1.0 | −1.0° | +15.0 |
| H. G., Artscl. (foot) | 1.14 | 1.80 | +0.66 | −1.0° | +14.0 |
| R. C., Artscl. (foot) | 2.7 | 6.8 | +4.1 | +4.5° | + 7.9 |
| J. D., Artscl. (foot) | 1.58 | 2.54 | +0.96 | +1.0° | +15.2 |
| W. A., Buerger's (foot) | 2.50 | 2.41 | −0.09 | 0.0° | + 8.8 |
| E. R.,* Buerger's (foot) | 7.8 | 7.4 | −0.40 | 0.0° | +16.3 |
| R. L., Buerger's (foot) | 1.94 | 4.84 | +2.9 | +2.2° | +30.0 |
| P. R., Raynaud's (hand) | 1.03 | 0.9 | −0.13 | −1.5° | +10.0 |
| H. G.,† Raynaud's (foot) | 1.5 | 1.85 | +0.35 | −1.6° | + 8.7 |
| S. L., Raynaud's phenomenon (hand) | 1.03 | 5.26 | +4.23 | +9.5° | + 9.3 |

*High basal flow thought to be due to insufficient basal observation. During the first 45 min. after glycine, blood flow fell to 4.1.

†One month postoperative bilateral dorsal sympathectomy. Hand flow taken at same time showed a decrease in blood flow. Patient has minimal symptoms in both lower extremities.

was 8.9 per cent and for the third 8.2 per cent. The over-all average increase for the three-hour period was 12.85 per cent (Fig. 2).

*Skin Temperature.*—Four patients had a rise in the skin temperature of the extremity opposite to the one in the plethysmograph (that is, great toe or index finger when foot or hand were used in the experiment). The smallest rise was 1.0° C., and the greatest 9.5° C. (Table I). Two patients demonstrated no appreciable change in temperature and four had a fall between 1.0 to 1.6° C. When a rise in temperature did occur, it came between 1½ to 3 hours after the administration of glycine.

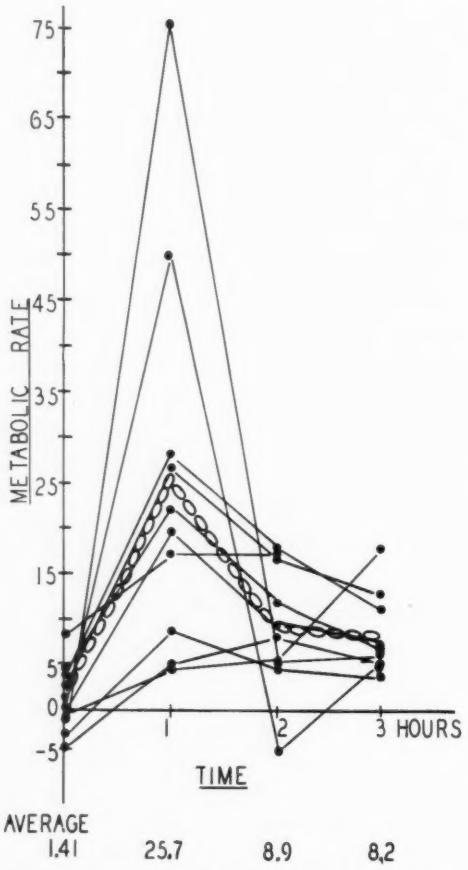


Fig. 2.—Oxygen consumption following the administration of 20 Gm. of glycine.

*Plethysmography.*—Seven of the ten patients in this experiment had an increase in blood flow which became apparent between 1 to 3 hours after the oral administration of glycine. The greatest rise usually occurred in the third hour. The range of increase was from 0.35 to 4.23 c.c. per 100 c.c. limb volume per minute (Table I). Two of these increases (0.35 and 0.66 c.c. per 100 c.c. limb per minute) could be interpreted as being changes within the limits of error of the venous occlusion method. (Figured in terms of per cent increase over

normal, however, these figures represented a 50 per cent and 20 per cent increase.)

Three patients demonstrated a decrease in blood flow ranging from 0.09 to 0.40 c.c. per 100 c.c. limb volume per minute, which again is probably within the limits of error of the method.

## CLINICAL STUDY

In recent years it has been the policy of this clinic to treat patients with peripheral vascular disease conservatively for a period of two to three months prior to definitive surgical therapy. This conservative program has consisted of abstinence from the use of tobacco, Buerger-Allen exercises, periodic rest and proper care of the affected member, local and systemic treatment of coexisting infection, and the use of various vasodilating agents, such as papaverine, alcohol, tetra-ethyl-ammonium compounds, lumbar sympathetic block, etc. This program is usually carried out on an outpatient basis, but in the more severe cases patients are hospitalized. The patients are followed carefully and required to report for re-examination at weekly or biweekly intervals for at least the first month. If satisfactory improvement is being made, the frequency of these visits is diminished, and eventually, when asymptomatic or improved to a status compatible with his normal activities, the patient is discharged on a modified conservative program. Patients who fail to improve, or whose improvement is not sufficient for their normal requirements, are hospitalized and either given further conservative care or treated surgically as indicated, that is, sympathectomy, peripheral neurectomy, amputation, etc.

A clinical study of the use of glycine in place of the vasodilating agent in the program of conservative therapy just outlined was conducted on sixty patients with peripheral vascular disease. Glycine was administered orally in doses of 20 Gm. two or three time daily. This amino acid dissolves readily in most liquids; in general, our patients preferred to mask its very sweet taste with some vehicle such as black coffee, grapefruit or lemon juice, or milk flavored with bitter chocolate. There were no untoward reactions from its use. A few individuals complained of nausea but this was avoided when the drug was taken after meals. Many patients noted a feeling of warmth in their hands and feet within one to two hours after taking glycine; this sensation usually persisted to some degree for five to seven hours. Glycine is relatively inexpensive; the use of alcohol, four ounces daily, costs more than twice as much.

Of the sixty patients studied, thirty-five had arteriosclerotic peripheral vascular disease, thirteen had thromboangiitis obliterans, seven had Raynaud's disease or Raynaud's phenomenon, and five had miscellaneous vascular disorders with associated vasospasm. All patients except the obvious failures were followed for at least three months. A preliminary report of the results follows.

*Arteriosclerotic Peripheral Vascular Disease.*—The average age of these patients was 60 years; twenty-nine were males, six were females. Two patients had a coexisting chronic thrombophlebitis and four were diabetic. For the purpose of this study they were classified into four groups on the basis of the

severity of the disease. Group I had symptoms of claudication with physical findings of diminished peripheral pulses and minimal to moderate color changes and atrophy. Group II had claudication, rest or night pain, absent pulses, and moderate to severe color changes and atrophy. Group III comprises those cases in which ulceration had occurred and in Group IV gangrenous changes were present.

The results of treatment in the arteriosclerotic group are presented in Table II. Those patients who are catalogued as having complete relief or marked improvement were asymptomatic under the conditions of their normal daily activity. Many of those in the slightly improved category have been able to return to their former occupations but still must limit their activity to some degree. As with any form of treatment of obliterative vascular disease, the more severe the arterial insufficiency, the poorer the end result. Three of the eight patients with peripheral gangrene had definite relief of pain, but in only one instance was this maintained. Of the entire group, thirteen patients were unimproved or had such temporary improvement that glycine therapy was abandoned in favor of intensive treatment with tetra-ethyl-ammonium. Ten of the thirteen were eventually advised to have a sympathectomy. The lesions healed in only two patients with ulceration and one with gangrene; however, there were no observed physical changes in patients with a less severe degree of vascular insufficiency.

TABLE II. ARTERIOSCLEROTIC PERIPHERAL VASCULAR DISEASE; RESULTS OF GLYCINE THERAPY

| GROUP | NUMBER OF CASES | RELIEF OF SYMPTOMS | | | | SUBSEQUENT TREATMENT | | |
|---|---|---|---|---|---|---|---|---|
| | | NONE | SLIGHT | MARKED | COMPLETE | HOSPITALIZED | T. E. A.* | SYMPATHECTOMY |
| I | 11 | 0 | 6 | 4 | 1 | 0 | 0 | 0 |
| II | 13 | 3 | 6 | 3 | 1 | 1 | 5 | 3 |
| III | 3 | 0 | 1 | 2 | 0 | 1 | 1 | 1 |
| IV | 8 | 5 | 1 | 2 | 0 | 7 | 7 | 6 |
| Total | 35 | 8 | 14 | 11 | 2 | 9 | 13 | 10 |

*Tetra-ethyl-ammonium compounds.

*Thromboangiitis Obliterans.*—Thirteen males with an average age of 38 years, on whom a diagnosis of thromboangiitis obliterans had been made, were treated with glycine. Six were of only moderate severity and were free of ulceration and gangrene; seven were classified as severe because of an open lesion and/or a loss of tissue had occurred. Our first few cases were unusually severe and the patients had been receiving intensive tetra-ethyl-ammonium therapy when this study was initiated. When this group became asymptomatic, they were discharged to take glycine as a maintenance measure. This proved to be successful and all but one of the patients in this study with severe thromboangiitis obliterans have been so treated (Table III). With one exception, the eight patients so managed have remained asymptomatic for periods of four to thirteen months. The one failure in this group had had bilateral lumbar sympathectomy and while under our care was most uncooperative about smoking, limitation of activity, and foot care.

Only five patients were treated with glycine alone; all had a sufficient degree of improvement so that they could return to work.

TABLE III. THROMBOANGIITIS OBLITERANS; RESULTS OF GLYCINE THERAPY

| CLASSIFICATION | NUMBER OF CASES | INITIAL T. E. A.* THERAPY | MAINTENANCE GLYCINE | GLYCINE THERAPY ALONE RELIEF OF SYMPTOMS | | | |
|---|---|---|---|---|---|---|---|
| | | | | NONE | MODERATE | MARKED | COMPLETE |
| Moderate | 6 | 2 | 2 | 0 | 1 | 2 | 1 |
| Severe | 7 | 6 | 6 | 0 | 0 | 1 | 0 |
| Total | 13 | 8 | 8 | 0 | 1 | 3 | 1 |

*Tetra-ethyl-ammonium compounds.

*Raynaud's Disease and Raynaud's Phenomenon.*—Five patients with Raynaud's disease and two with secondary Raynaud's phenomenon were treated with glycine alone (Table IV). Of the two patients who have experienced no improvement, one had previously been sympathectomized without significant benefit; the other patient was interested in treatment only if we would guarantee 100 per cent success. She was urged to try glycine without a guarantee and reported no change after a two-week period. Only one patient had complete freedom from attacks while on glycine; the usual improvement was in a diminution in the number and severity of the attacks plus a definite increase in the tolerance of cold.

TABLE IV. RAYNAUD'S DISEASE OR PHENOMENON; RESULTS OF GLYCINE THERAPY

| CLASSIFICATION | NUMBER OF CASES | RELIEF OF SYMPTOMS | | | PREVIOUS TREATMENT |
|---|---|---|---|---|---|
| | | NONE | MODERATE | COMPLETE | |
| Raynaud's disease | 5 | 2 | 2 | 1 | 1* |
| Raynaud's phenomenon† | 2 | 0 | 2 | 0 | 0 |
| | 7 | 2 | 4 | 1 | 1 |

*Bilateral Dorsal Sympathectomy.
†Secondary to (1) ruptured cervical disc and (2) arsenical intoxication.

*Miscellaneous.*—Vasospasm with associated cold sensitivity secondary to some other vascular disturbance such as chronic lymphedema, chronic thrombophlebitis, frostbite, and trench foot was somewhat ameliorated by the use of glycine. In two instances there was a dramatic response; in the other three cases, while the physical evidence of vasospasm disappeared, the patient was still symptomatic and required definitive therapy of the primary disease for eventual relief.

## DISCUSSION

The treatment of vascular disease is notoriously difficult to evaluate. Changes in the weather, activity of the patient, and exacerbations and remissions characteristic of the disease all serve to confuse the result. Because of the seriousness of the loss of limb, every effort is made to conserve the extremity. Under these circumstances combined therapy becomes the rule and further obscures the evaluation of new forms of management. We have attempted to reduce these factors to a minimum and have outlined the scope of our experimental and clinical study in the preceding paragraphs. While it may be argued that some of our patients would have experienced as much relief with only cessation of smoking and routine foot hygiene, we have now had over one year's

experience with this drug and believe that it is a definite adjunct to the conservative treatment of peripheral vascular insufficiency. Experimentally, glycine produces vasodilatation, certainly not to the degree encountered with the use of tetra-ethyl-ammonium or procaine sympathetic blockade,[13] but of a greater magnitude than has been reported for alcohol.[14, 15, 16] However, it is inexpensive, has none of the socio-economic disadvantages of alcohol, can be taken orally, is completely safe, and, theoretically, could produce virtually constant vasodilatation twenty-four hours a day.

## SUMMARY

1. The changes in blood flow, skin temperature, and oxygen consumption following the ingestion of the amino acid glycine were studied in ten patients with peripheral vascular disease.

2. The results of the conservative treatment of sixty patients with various peripheral vascular disturbances utilizing glycine as the vasodilating agent are presented.

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# AMPUTATION FOR GANGRENE DUE TO OCCLUSIVE ARTERIAL DISEASE

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THE incidence of gangrene due to occlusive arterial disease has been reduced by our greater knowledge of the peripheral circulation and earlier recognition and treatment. But in spite of this, our increasingly large diabetic population and greater longevity make the problem an ever-present and serious one in the older age groups.

The mortality following amputations for gangrene in these cases is still greater than that for most major surgical procedures in similar age groups. The published statistics indicate mortality rates varying from 39.1 per cent[1] to 10 per cent[2] and 4.7 per cent[3] for all cases. In the presence of cellulitis and rapid spreading gangrene the mortality may be as high as 66.7 per cent[4] and 51 per cent.[5] These figures indicate the seriousness of the problem and suggest the need for further study and discussion of methods to improve it.

## ANALYSIS OF CASES

This report is based on experience with 165 amputations for gangrene due to occlusive arterial disease performed by the surgical staff of the Michael Reese Hospital from 1936 to 1947 (Table I). The *hospital* mortality for the 165 amputations was 17.5 per cent. In the group there were 143 major amputations with a mortality of 19.5 per cent. The rate varied from a 25 per cent mortality in the 1936 to 1938 period before the use of sulfonamides to 15.5 per cent in the 1939 to 1944 period when the sulfonamides were used, and 11.7 per cent in the 1945 to 1947 period during which penicillin was used. All the hospital deaths regardless of their relation to the amputations were included in these statistics. Actually, of the total of 28 deaths, 6 were not related to the amputation. In 5 cases death was the result of a pre-existing cardiac condition 28, 31, 50, 54, and 74 days after amputation and long after the operative wound had healed, and 1 died of a gastric hemorrhage on the twenty-first day of an otherwise uneventful convalescence. Exclusion of these deaths from the statistics results in an *operative* mortality rate of 13.3 per cent for the 165 amputations, 15.3 per cent for the major amputations, 8.5 per cent for all the amputations in the 1945 to 1947 period, and 9.6 per cent for the major amputations in that period (Table II).

The decline in the mortality rate over the years seems to be due in great part to the decrease in the incidence of infection and sepsis (Table VI) and coincides with the use of the antibiotics and blood transfusion.

There were 97 amputations in diabetic patients with a hospital mortality of 13.3 per cent, 47 cases of senile arteriosclerosis with a mortality of 20.9

Received for publication, July 26, 1948.

TABLE I. AMPUTATIONS AND HOSPITAL MORTALITY IN VARIOUS PERIODS

| YEARS | MAJOR AMPUT. | HOSP. MORT. | PER CENT | TOTAL AMPUT. | HOSP. MORT. | PER CENT | REMARKS |
|---|---|---|---|---|---|---|---|
| 1945-1947 | 31 | 4 | 12.9 | 35 | 4 | 11.7 | Penicillin |
| 1939-1944 | 78 | 14 | 17.9 | 90 | 14 | 15.5 | Sulfonamides, blood |
| 1936-1938 | 34 | 10 | 29.4 | 40 | 10 | 25.0 | |
| Total | 143 | 28 | 19.5 | 165 | 28 | 17.5 | |

TABLE II. OPERATIVE MORTALITY (HOSPITAL MORTALITY CORRECTED)

| AMPUTATIONS | YEARS | NO. | OPERATIVE DEATHS | PER CENT |
|---|---|---|---|---|
| Total | 1936-1947 | 165 | 22 | 13.3 |
| Major | 1936-1947 | 143 | 22 | 15.3 |
| Total | 1945-1947 | 35 | 3 | 8.5 |
| Major | 1945-1947 | 31 | 3 | 9.6 |

per cent, 14 cases of thromboangiitis obliterans with a 7.1 per cent mortality, and 7 cases of arterial embolism with a 57.1 per cent mortality (Table III). The associated severe cardiac disturbance in the cases of peripheral arterial embolism was a contributing factor in that mortality and the greater average age of the senile arteriosclerotic patients was a factor in that group. Our diabetic patients were adequately prepared and controlled during the postoperative period with intravenous glucose and regular insulin in most instances. In those patients who were operated upon under refrigeration anesthesia the dietary control was no problem and they received their regular diets and insulin even on the day of surgery. Contrary to the reported statistics our mortality in this group of diabetics was fairly low (13.3 per cent). There was a greater incidence of wound infection in these patients and for this reason it seems that in the diabetic patients especially intensive pre- and postoperative treatment with antibiotics is important.

TABLE III. CAUSES OF THE GANGRENE

| CAUSE | MAJOR AMPUT. | HOSP. MORT. | PER CENT | MINOR AMPUT. | TOTAL AMPUT. | HOSP. MORT. | PER CENT |
|---|---|---|---|---|---|---|---|
| Diabetic endarteritis | 83 | 13 | 15.6 | 14 | 97 | 13 | 13.3 |
| Arteriosclerosis | 44 | 10 | 22.9 | 3 | 47 | 10 | 20.9 |
| Thromboangiitis obliterans | 9 | 1 | 11.1 | 5 | 14 | 1 | 7.1 |
| Emboli | 7 | 4 | 57.1 | 0 | 7 | 4 | 57.1 |
| Total | 143 | 28 | 19.5 | 22 | 165 | 28 | 17.5 |

There were 124 amputations above the knee, 19 below the knee, 6 through the foot, and 16 toe amputations (Table IV). Although all the deaths followed amputations above the knee, we believe that it was purely coincidental and not cause and effect. The lower amputations were all in better risk patients.

TABLE IV. TYPE OF AMPUTATION

| TYPE | NO. | HOSP. MORT. | PER CENT |
|---|---|---|---|
| Thigh | 124 | 28 | 22.5 |
| Leg | 19 | 0 | 0 |
| Foot | 6 | 0 | 0 |
| Toe | 16 | 0 | 0 |

Of the 22 minor amputations (Table V), 10 later required a major reamputation (45.5 per cent). There were 14 diabetic cases with 7 reamputations (50 per cent), 3 cases of senile arteriosclerosis with 2 reamputations (66 per cent), and 5 cases of thromboangiitis obliterans with 1 reamputation (20 per cent). In the cases of diabetic and senile arteriosclerosis, minor amputations were successful only in the presence of a pulsating vessel at the ankle or after a preliminary sympathectomy. In several young men with thromboangiitis obliterans, amputation of a toe was successful in the absence of such a pulse, probably because of the better development of collaterals in that condition. From our experience we believe that it is wise to precede all minor amputations by a sympathetic ganglionectomy.

TABLE V. MINOR AMPUTATIONS AND RE-AMPUTATIONS

| CAUSE | MINOR AMPUT. | MAJOR REAMPUT. | PER CENT |
|---|---|---|---|
| Diabetic endarteritis | 14 | 7 | 50.0 |
| Arteriosclerosis | 3 | 2 | 66.6 |
| Thromboangiitis obliterans | 5 | 1 | 20.0 |
| Emboli | 0 | 0 | 0 |
| Total | 22 | 10 | 45.4 |

1 leg amputation later required reamputation above the knee.
Autoamputations of toes are not included in table.

Ten of the 28 hospital deaths (Table VI) were due to cardiovascular diseases as cerebral hemorrhage and thrombosis, coronary occlusion, auricular fibrillation, and cardiac decompensation. In most instances the cardiac condition existed prior to the onset of the gangrene and in 4 of the deaths which followed arterial embolism it was the indirect cause of the gangrene. There were 14 deaths due to pneumonia, wound sepsis, pyelonephritis, and decubitus ulcer. Of these, 13 occurred before the advent of penicillin. There were 2 deaths due to pulmonary embolism, 1 due to gas gangrene, and 1 due to a gastric hemorrhage. As indicated in Table VI, deaths due to sepsis have been reduced greatly since the use of antibiotics but the cardiovascular system continues to be a great hazard. The average age of the 28 patients who died was 66.5 years.

TABLE VI. CAUSES OF DEATH

| YEAR | TOTAL AMPUT. | HOSP. MORT. | AVER. AGE | CARDIOV. DISEASE | SEPSIS PNEUMONIA | EMBOLI | GAS GANGRENE | GASTRIC HEMORRHAGE |
|---|---|---|---|---|---|---|---|---|
| 1947 | 16 | 2 | | 2 | | | | |
| 1946 | 16 | 2 | | 1 | 1 | | | |
| 1945 | 3 | 0 | | | | | | |
| 1944 | 5 | 2 | | | 2 | | | |
| 1943 | 15 | 2 | | 2 | | | | |
| 1942 | 33 | 8 | | 3 | 4 | 1 | | |
| 1941 | 23 | 0 | | | | | | |
| 1940 | 10 | 2 | | 1 | 1 | | | |
| 1939 | 4 | 0 | | | | | | |
| 1938 | 11 | 6 | | 1 | 3 | 1 | 1 | |
| 1937 | 17 | 4 | | | 3 | | | 1 |
| 1936 | 12 | 0 | | | | | | |
| Total | 165 | 28 | 66½ yr. | 10 | 14 | 2 | 1 | 1 |

The 1 death from gas gangrene occurred in 1938 following an emergency amputation because of high fever and spreading cellulitis. No antitoxin was administered. The patient died the following day. The gas bacillus infection apparently was present on admission. It is our policy to administer a therapeutic dose of tetanus and gas gangrene antitoxin on the day before amputation.

During the same period, 1936 to 1947, 22 major amputations were performed for nongangrenous conditions such as sarcoma, osteomyelitis, etc (Table VII). These patients averaged 30 years younger than the ones with amputations for gangrene. There was only 1 hospital death (4.5 per cent).

TABLE VII. AMPUTATIONS FOR OTHER CONDITIONS, 1936-1947

| CAUSE | NO. | MORT. |
|---|---|---|
| Chronic osteomyelitis | 5 | |
| Compound fracture with osteomyelitis | 6 | |
| Sarcoma | 8 | 1* |
| Arthroplasty | 1 | |
| Gunshot | 1 | |
| Congenital deformity | 1 | |
| Total | 22 | 1 (4.5%) |

*A 55-year-old man, who died on the fortieth day of sepsis and pneumonia.

Preliminary ligation of the femoral vein on the side of the amputation was carried out in 34 of our cases as recommended by Veal[6] as prophylaxis against pulmonary embolism. Only one of these patients died of a pulmonary embolus which on post-mortem examination was found to come from the opposite extremity. There was no edema of the stump in any of our cases as a result of this ligation, no increased bleeding, and no delay in healing. The number of vein ligations is too small for generalizations to be made regarding the efficacy of this procedure in preventing embolization, but it seems quite logical. After the usual amputation either below or above the knee, a considerable length of the popliteal and femoral veins remains as a "dead end" in which thrombosis occurs and emboli may originate. Ligation of the femoral vein below the profunda just before the amputation eliminates this and adds very little to the operating time or shock.

One hundred seventeen amputations were performed under general inhalation anesthesia, 23 under spinal anesthesia, 22 under refrigeration, and 3 under nerve block anesthesia. We found that even our elderly patients tolerated inhalation or spinal anesthesia well. Refrigeration anesthesia[7] was used since 1941 in the acutely ill and debilitated individuals and in the poor risk diabetic patients in whom the dietetic management was a problem. Thirty-eight amputations were attempted under refrigeration anesthesia but in only 22 cases was it entirely successful. In all the others it had to be supplemented with an inhalation anesthetic when the nerves were clamped and the bone severed. There was practically no shock following amputations under refrigeration and most of the patients were able to return to the regular dietetic management the same day. There was a slight increase in the

serous drainage from the wound and some delay in healing. In 2 of our earliest cases there was some sloughing of the skin edges. Our method of refrigeration of an extremity for anesthesia is described elsewhere.[8]

In addition to its use as an anesthetic agent, refrigeration and a tourniquet have been used in a large number of our patients prior to surgery as a means of combating the severe pain and cellulitis present. By these means we were able to produce, in effect, a physiologic amputation for several days before actual removal of the limb. This enabled us to carry out adequate preoperative measures. Many of our severely ill, toxic, and debilitated patients improved so much because of refrigeration that the amputation was changed from one of emergency in a poor risk patient to an elective procedure in a well-prepared individual. It is our opinion that refrigeration, both as a preoperative measure and as an anesthetic, has aided materially in lowering the amputation mortality.

## DISCUSSION

Gangrene of an extremity is the end result of progressive peripheral vascular insufficiency which has not responded to any of our methods of treatment. In dealing with this problem the surgeon must realize and take into account the fact that most of these patients have involvement of the whole vascular tree including the coronaries and the cerebral vessels, as well as the extremities. His first objective is to preserve the life of the patient, and the second to limit the gangrene and amputate at such a level as will give the individual the best possible walking limb when fitted with a prosthesis. In these days of antibiotics, blood transfusion, and refrigeration, gangrene is seldom an emergency, and the surgeon has adequate time to evaluate the patient carefully and to carry out measures that will aid possible autoamputation or permit amputation at a low level.

As a rule most of these elderly patients are debilitated from long suffering and infection by the time they come to surgery. It is of utmost importance that the malnutrition, avitaminosis, dehydration, and hypoproteinemia be corrected by appropriate measures including parenteral fluids, plasma or hydrolysates, and vitamins. As in all surgical procedures this will materially lower the mortality and morbidity following amputation. The secondary anemia may be very severe in some of the patients on admission to the hospital. We believe that it is much wiser to correct this quickly by blood transfusion than to wait for improvement with diet, iron, etc. This results in a proportional increase in the nourishment and oxygen reaching the extremity, and one is frequently amazed by the marked local improvement and relief of pain that follows along with the general increased strength of the patient.

A high percentage of the diabetic and senile arteriosclerotic patients have some form of heart disease. It goes without saying that all possible measures to improve the cardiac status should be carried out during the preoperative period. An anesthetic with a high oxygen concentration or refrigeration should be used and the patient should be placed in an oxygen tent immediately after surgery. Because of the ever-present infection in gangrene

and the high incidence of gas bacillus infection that is reported following amputation,[2] a course of antibiotic therapy and a prophylactic therapeutic dose of gas gangrene and tetanus antitoxin should be given to every patient before surgery.

Various methods have been recommended to determine the best level for amputation, such as the histamine, the fluorescin, and reactive hyperemia tests, and the skin temperature determination. We have found the oscillometric reading to be the most reliable for this purpose. The presence of a pulsating vessel at any joint, however slight it may be, is an indication that it is reasonably safe to amputate just below it. In young patients with gangrene due to thromboangiitis obliterans we have been fortunate occasionally to obtain a healed wound in the absence of a pulsating artery at the joint proximal to the site of amputation when the skin temperature was within normal limits. In those younger individuals who must perform active work for a livelihood and who are in reasonably good general condition it is often worth while risking a possible reamputation in order to save an ankle or a knee. But in the elderly and debilitated and in those with a rapid spreading cellulitis and gangrene such conservatism and risk are not warranted.

Pain due to an ulcer or gangrene of a toe usually subsides with improvement in the local inflammation and in the circulation brought about by medical means or sympathectomy. When it persists, peripheral nerve section or anesthetization with procaine in oil is helpful.[9, 10] In addition to relieving the pain these procedures often cause sufficient vasodilation to heal the lesion. In a high percentage of such cases, relief of the pain by these means and continuation of conservative treatment have resulted in healing of ulcers and autoamputation of toes.

In the absence of cellulitis or of spreading gangrene we have favored conservatism in such cases for a reasonable length of time. However, during this period there is constant danger of a spreading infection which must be avoided by all possible means. Frequent short courses of antibiotics and small blood transfusions are especially helpful.

The most common paths of infection from a gangrenous toe are along the tendons and in the fascial spaces. The surgeon frequently and advertently initiates such a complication by overzealous débridement of the gangrenous part. Often a gangrenous phalanx is removed before the attached tendon has become fixed by the inflammation, thus allowing it to retract toward the muscle belly carrying infectious material. When such a spread of infection and localized suppuration does occur, the treatment should be along recognized surgical principles as incision and drainage. We have found conservatism and delay in removing gangrenous tissue to be the safest.

Amputation of a toe can be performed in most instances under nerve block anesthesia. Any or all of the five nerves supplying the foot, the posterior tibial, the superficial and deep peroneals, the sural, and the saphenous nerves can be anesthetized above the ankle and complete anesthesia of the operative field obtained. After adequate skin preparation, an elliptical incision is made about the base of the involved toe and extended upward on the

dorsum of the foot to permit removal of the head of the adjacent metatarsal. The tendons and bone are separated proximal to the gangrenous area and the toe is removed from proximal distally to prevent contamination of the fresh wound surfaces. The wound edges are brought together loosely with silk.

The transmetatarsal amputation is best performed under general or spinal anesthesia. The surgeon should plan a fairly long plantar skin flap because the dorsal skin tends to retract and to heal poorly. The metatarsals are severed at the junction of the proximal and middle thirds and the bleeding points ligated with the finest possible catgut or silk. The skin should be closed loosely over the end of the bones and a small Penrose drain led out between the sutures for several days.

Of the major amputations, one below the knee is the most preferable. The walking ability of a patient with such an amputation and a properly fitted prosthesis is so much superior to that with a supracondylar amputation that the surgeon should feel obligated to perform the lower amputation whenever possible. Most of our patients who have had such amputations have been able to return to gainful occupations. There is usually very little difficulty in healing of the stump in the presence of even a weakly pulsating popliteal artery, and in a few of our younger patients we have obtained well-healed stumps even in the absence of such a pulsation but with normal skin temperature to the ankle. Ordinarily occlusion of the femoral artery is an indication for a supracondylar amputation.

Recently we have performed lumbar sympathetic ganglionectomy in some of our cases when the popliteal and femoral pulsations were absent in order to improve the circulation in the leg so that a low amputation could be performed with less risk of nonhealing. Of 10 patients so treated we were able to save the ankle in 2 and the knee in 7. In each case the clinical examination indicated a supracondylar amputation as the one of choice before the sympathectomy. In the tenth patient the circulation below the knee was not improved sufficiently to warrant a low amputation. These results seem to us to recommend preliminary sympathetic ganglionectomy whenever possible in order to perform a low amputation. Although this seems like a radical means of preparation for amputation, we should realize that a useful knee may mean the difference between a patient who can walk well and earn a livelihood and one who cannot. In the individual who will have to be on his feet this procedure should be considered seriously even though there is the added risk of a second operation.

In all our major amputations we planned for side-bearing stumps. Although it is generally agreed that an end-bearing stump such as in the Pirogoff and Gritti-Stokes operations is desirable whenever possible, the very nature of the conditions for which these amputations were performed made the risk of nonfusion of the bones too great for the possible advantages. The optimum site for amputation below the knee is through the middle third of the leg in order to allow ample clearance for the knee and ankle mechanisms of the prosthesis, but if necessary a bone stump as short as 1 inch below the tibial tubercle can be fitted with an adequate prosthesis.

A skin incision is made 6 to 7 inches below the tibial tubercle so shaped as to make a slightly curved anterior and a somewhat longer posterior flap. On the lateral surface the incision is extended upward for 2 inches to permit better exposure of the fibula. The muscle compartments are opened about 1½ to 2 inches proximal to the skin incision and the major blood vessels and nerves are exposed, clamped separately, and cut. The muscles and the interosseous membrane are then severed at that level and the tibia cut with a handsaw at the point of greatest muscle retraction, or approximately 4 inches below the tibial tubercle. The crest of the tibia is beveled to eliminate the sharp angle which often results in pain and skin necrosis. The fibula is then exposed through the lateral incision and removed at a point 1 to 2 inches above the tibial level. The vessels and nerves are then ligated. The periosteum is not removed from the bone ends. The investing fascia of the leg is sutured and the skin closed with interrupted sutures. A rubber drain is inserted below the fascia. The skin should cover the stump very loosely. Skin traction is applied immediately by means of adherent and stockinet or adhesive tape to obtain a painless stump and better healing, and is maintained until the wound is healed. We do not use a tourniquet for any amputation.

Above the knee we prefer the modified guillotine supracondylar amputation. Slightly rounded anterior and posterior skin flaps of equal length are fashioned extending to the patella. The skin is allowed to retract and the fascia and muscle bundles are severed at the point of greatest skin retraction. The major vessels and the sciatic nerve are then isolated, clamped separately, and cut. The bone is severed with a handsaw at the point of greatest muscle retraction, leaving the periosteum attached up to the cut end. The vessels and the nerve are then ligated. A purse-string suture through the fascia will cover the stump of the femur and center it between the muscles. The skin is then sutured loosely. A Penrose drain is placed between the fascia and skin and led out between the sutures or through a posterior stab incision. We have found that skin traction is unnecessary here if the flaps are of adequate size, but when there is any tension on the skin, traction is applied immediately. In some of our cases the sciatic nerve end was injected with alcohol and not in others. The results were the same. There was not one known painful neuroma in the series.

Careful attention to details during the early postoperative period will reduce the mortality and morbidity. Most of our patients are given a transfusion of 500 c.c. of blood either during or immediately after operation to overcome blood loss and shock. The older individuals cannot stand much blood loss without danger. Antibiotics are administered subcutaneously to all for the first five to seven days. Our older patients and those with cardiorenal or pulmonary diseases are placed in an oxygen tent immediately on returning to bed. We believe that this has been a great aid in tiding some over the critical period. Deep breathing and well leg exercises are usually started on the first day after operation, and in most cases unless otherwise contraindicated the patient is placed in a wheel chair on the second or third

day. Application of an ice bag to the stump over the dressings for short periods frequently relieves postoperative pain, but when necessary morphine is used.

In a few of our patients we have noticed a brief period of mental derangement in the early postoperative period with complete loss of memory for recent events. Usually this cleared up spontaneously in a matter of days.

## SUMMARY

1. Results and experiences with 165 amputations for gangrene due to peripheral arterial disease at the Michael Reese Hospital are presented.

2. The total *hospital* mortality for these cases was 17.5 per cent and was reduced from 25 per cent in 1936 to 1938 to 11.7 per cent in 1945 to 1947. Elimination of the hospital deaths due to conditions which existed prior to the amputation and which occurred after the wound had healed resulted in an *operative* mortality of 13.3 per cent for the 165 amputations (1936 to 1947) and 8.5 per cent for the 1945 to 1947 period.

3. The improved results are due in great part to better pre- and postoperative care, blood transfusions, antibiotics, and refrigeration.

4. Refrigeration reduces absorption of toxins from the area of gangrene or inflammation and permits adequate preoperative preparation. When used as an anesthetic agent it prevents shock.

5. Preliminary sympathetic ganglionectomy in selected cases will permit amputation at a lower level than would be possible otherwise.

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# EXPERIMENTAL ANASTOMOSES OF VESSELS TO THE HEART

## Possible Application to Superior Vena Caval Obstruction

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SEVERE obstruction of the superior vena cava, in man, causes marked elevation of the venous pressure in the head, neck, and upper extremities, and often results in edema of the upper body, and pleural effusion. If the patient survives, profound disability often results because, though an abundant collateral circulation develops, this seldom is sufficient to compensate for the obstruction. Although the obstruction is due most commonly to aneurysm or mediastinal tumors, there are many cases for which various types of chronic fibrosing mediastinitis are responsible.[1, 2, 3] The correction of such fibrous constriction is frequently impossible and, therefore, relief of congestion requires the establishment of an alternative venous route to the heart.

Carlson's[4] experimental observations in the dog led him to conclude that acute occlusion of the superior vena cava below the azygos vein was fatal, but in two out of three trials he succeeded in producing a two-stage obstruction of the superior vena cava and the azygos vein. Six out of seven of his dogs with an acute occlusion above the junction of the azygos survived.

It has been demonstrated amply that arterial shunts will remain patent within the thorax,[5, 6] and quite recently Hanlon and Blalock,[7] in seeking a method for the treatment of transposition of the aorta, demonstrated that under experimental conditions an anastomosis of the right pulmonary vein to the superior vena cava was technically feasible, though pulmonary vein to auricle anastomoses were unsatisfactory.

The first of our experiments, done in September, 1947, dealt with utilizing the azygos vein to shunt around an obstruction in the superior vena cava. Some success with these anastomoses[8] led to a series of experiments in which subsequently the superior vena cava was anastomosed to the right auricle to relieve chronic as well as acute superior vena caval obstruction.

### GENERAL PROCEDURE

The experiments were conducted on adult dogs weighing 11 to 18 kilograms. They were anesthetized with veterinary nembutal (1 c.c., or 5 gr., for each 5 pounds body weight given intravenously). In a few instances this was supplemented with inhalation ether. A mechanical respirator and endotracheal tube were used. The thorax was entered through the right fourth interspace, it being possible to gain enough exposure without rib resection. During the vascular suture, a bloodless field was maintained by using Blalock or bulldog clamps on the veins, and a special rubber-covered clamp on the auricle. With two exceptions, the anastomoses were accomplished with the continuous everting mattress type of suture, using 00000 arterial silk on a No. 9 minimum-trauma

Received for publication, July 15, 1948.

needle. About 1 c.c. of heparin solution was used at the anastomotic site in each of the experiments, but no anticoagulants were administered postoperatively. To complete the procedure 100,000 units of penicillin were instilled into the pleural space, the ribs were approximated with two linen sutures, and the various other layers with interrupted silk. Jugular venous pressures were taken pre- and postoperatively by the direct method. In some instances direct caval pressures were taken by means of cardiac catheters passed through the

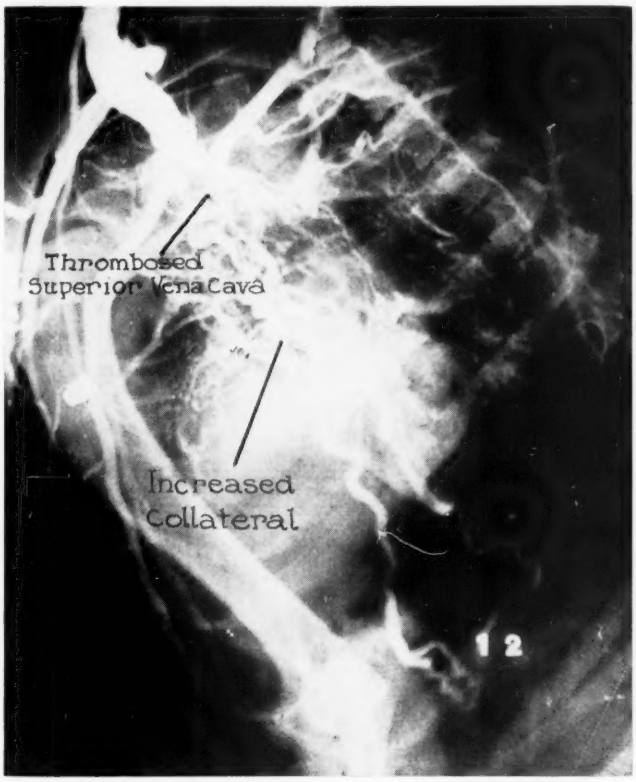


Fig. 1.—Diodrast angiogram in a dog with complete thrombosis of the superior vena cava extending to the subclavian vein, of at least three weeks' duration. This followed several operative procedures including ligation below the level of the azygos vein, and an unsuccessful atriocaval anastomosis. Compare with Fig. 9, in which identical procedures were performed, but the venous congestion was relieved by thrombectomy and atriocaval shunt.

brachial vein. Patency of anastomoses was determined by angiography,* thoracotomy, or necropsy. Electrocardiograms were taken preoperatively in two of the animals which had atriocaval anastomoses, and postoperatively on five animals in this group.†

### ACUTE THROMBOSIS OF THE SUPERIOR VENA CAVA

Following partial ligation or anastomosis, acute thrombosis of the superior vena cava occurred four times. These animals appeared listless, had cyanosis of the tongue, and distention of the jugular veins. There was usually edema of

*The angiograms were done under the direction of Dr. Charles E. Duisenberg of the Department of Roentgenology.

†The expression atriocaval anastomosis is used to indicate a shunt between the superior vena cava and the right auricular appendage.

the head, neck, and shoulders, in some instances involving the chest wall. The jugular venous pressures measured by the direct method rose to above 20 cm. of water, the average in these animals being 22 cm. compared with a normal of 7 cm., the zero point being 5 cm. above the table with the animal lying on his left side. The increased venous pressure caused a transudation of fluid into the pleural space, which was always enough to cause respiratory embarrassment (Fig. 1).

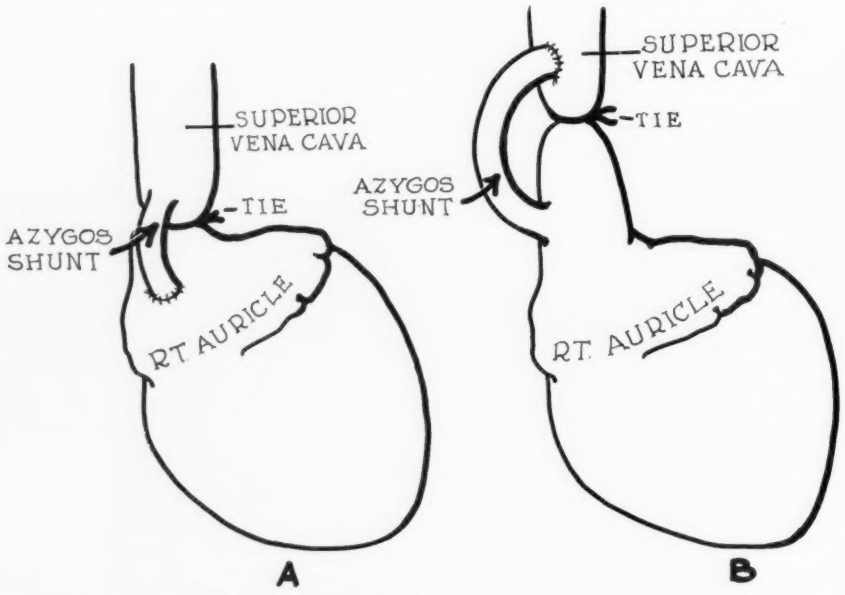


Fig. 2.—*A*, This type of shunt was done two to four weeks after partial ligation of the superior vena cava. The occlusion was made complete at the time of the shunt. *B*, Method of performing a one-stage obstruction of the superior vena cava and utilizing the azygos vein as a shunt.

## AZYGOS VEIN SHUNTS

These experiments were conducted to determine whether the azygos vein could be used to circumvent an obstruction in the superior vena cava. In two animals the superior vena cava was obstructed completely above the level of the azygos vein by means of a braided silk ligature. At the same procedure the azygos vein was freed from the thoracic wall and anastomosed to the superior vena cava above the point of obstruction (Fig. 2, *B*). Both animals survived this procedure without showing any of the usual effects of complete superior vena caval occlusion. One of the animals was killed after one week, and the other after 180 days, necropsy in both cases revealing a smooth, open stoma which had remained approximately its original size (Fig. 3).

In another series of six animals an incomplete obstruction of the superior vena cava was produced, just below the level of the azygos vein, by loosely tying a braided silk ligature around it. This raised the jugular venous pressure to two or three times the normal as measured by the direct method. Five animals survived this procedure, the sixth dying from a chylothorax caused by injuring the thoracic duct. It was hoped that the increased load carried by the azygos vein might cause it to dilate and become a more suitable size for anastomosis,

but very little dilatation occurred. Perhaps a longer interval would have allowed more dilatation. Two to four weeks later these animals were again subjected to thoracotomy; the superior vena cava was completely occluded with another ligature at the same level (below the azygos), and the azygos vein was shunted into the right auricle to bridge the obstruction (Fig. 2, *A*). In order to perform this anastomosis, a rubber-covered clamp was applied to the auricle between the superior and inferior vena cavae, temporarily pinching off a small segment of it. A small circular piece of atrial wall, the size of the azygos stoma, was excised with small curved scissors to provide an opening into the atrium. In two of these five animals the postoperative venous pressures remained low, and at thoracotomy after 82 days in one instance, and necropsy after 114 days in

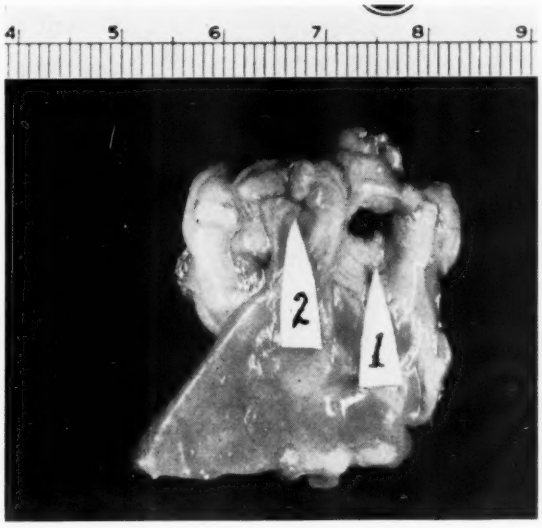


Fig. 3.—(*1*) Azygos vein to superior vena cava anastomosis of 180 days' duration. (2) Site of obstruction in the superior vena cava produced by a braided silk ligature.

the other, patent, though somewhat narrowed, anastomoses were found (Fig. 4). In two other animals the venous pressures indicated patent stomas in the immediate postoperative period, but in a few weeks the pressures became elevated and angiograms revealed occlusion at the site of anastomosis. These findings were confirmed by thoracotomy, at which fibrous occlusion of the anastomosis in both instances was found. Curiously enough, in neither animal had the azygos vein become thrombosed. The fifth animal in this group developed acute thrombosis of the superior vena cava on the fourteenth day after the azygos shunt.

### ATRIOCAVAL ANASTOMOSIS

In these experiments the superior vena cava was transected just above its junction with the azygos vein and anastomosed to the right auricular appendage. There were ten animals in this group. Five had had a preliminary partial ligation of the superior vena cava below the azygos vein.* In the remaining five animals the anastomosis was accomplished without preliminary ligation.

*In three of these five dogs an azygos to atrial shunt had also been done, but the stoma had become occluded.

The operative procedure was as follows: The superior vena cava was mobilized by incising the pleura along its margins, permitting the separation of the tissues from its posterior wall. The pericardium ascends for several centimeters on the lower end of the superior vena cava. Its sac was incised over the lower end of the superior vena cava allowing the upward reflection of pericardium to be cleared from the vein by sharp dissection. This incision, when carried downward, exposed the right auricle. A few cubic centimeters of 1 per cent procaine hydrochloride were applied to the surface of the auricle. The lower end of the superior vena cava, just above the level of the azygos vein,

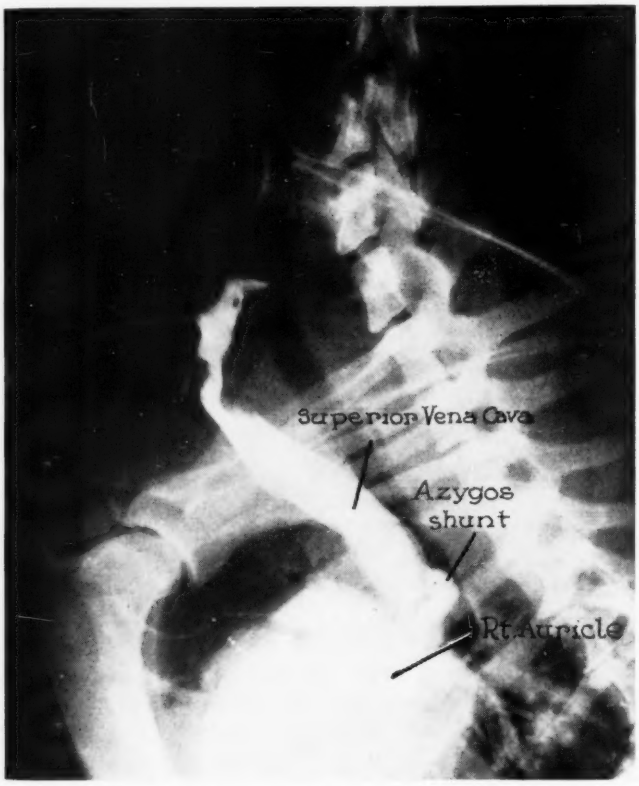


Fig. 4.—Angiogram showing an azygos vein to right auricle shunt of three weeks' duration.

was clamped with two special hemostats (Fig. 5). About 3 cm. above this a rubber-covered clamp was applied, and the superior vena cava was transected just above the hemostats. The auricular appendage was then picked up with toothed forceps, a rubber-covered clamp was applied across its base, and a small piece of the tip of the auricular appendage was excised with scissors. A few extrasystoles occur as the auricle is handled, but these cease in a few seconds, as do contractions in that portion of the auricle beyond the clamp. The procaine probably minimizes the number of extrasystoles. The end of the superior vena cava was then anastomosed to the opening in the auricle, using a continuous everting silk suture. It is a little difficult to evert the irregular musculature of the auricular wall, but this can be accomplished if care is taken. A few in-

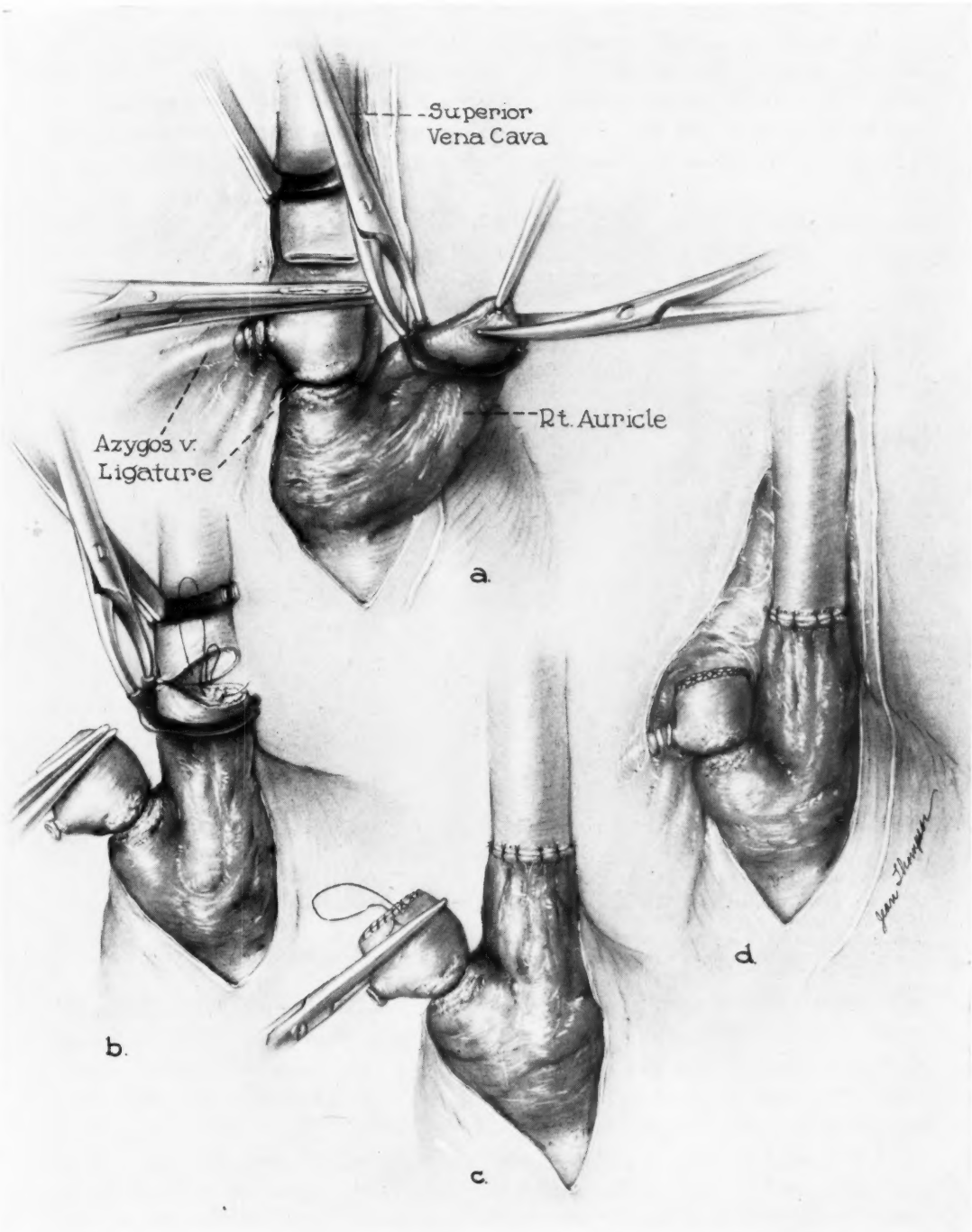


Fig. 5.—Technique of anastomosing the superior vena cava to the right auricle.

*a*, The superior vena cava has been transected above the level of the azygos vein; the right auricular appendage is grasped with a rubber-covered clamp while the tip is excised, thus creating a lumen into the atrium.

*b*, A continuous everting suture of arterial silk is used. A broad cuff is everted so that none of the cut edge of the auricular muscle remains in the lumen.

*c*, The anterior row is reinforced with a few interrupted sutures. The stump of the superior vena cava is closed with a double row of arterial silk.

*d*, The completed anastomosis.

terrupted sutures were added to the anterior row. There was no severe bleeding when the clamps were removed, and only two anastomoses required an additional suture. The stump of the transected superior vena cava was closed with a continuous arterial suture. In all but two animals the azygos vein was occluded at this point. The pericardium was approximated over the anastomosis with several interrupted silk sutures.

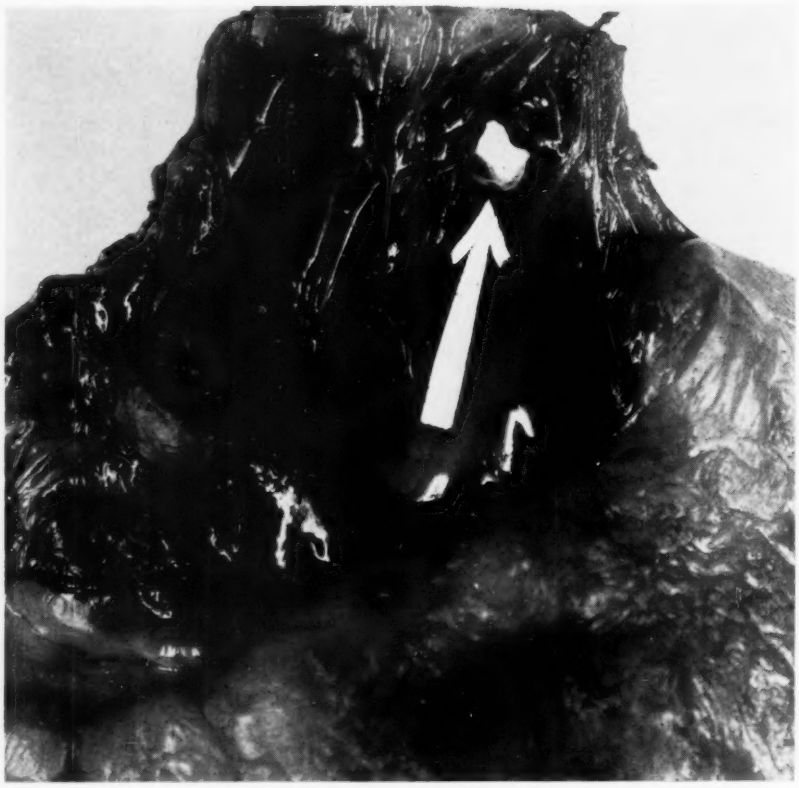

Fig. 6.—Superior vena cava to right auricle shunt of thirty-five days' duration. A No. 24 bougie passed through this stoma, while the cava admitted a No. 32.

There were no operative deaths following this procedure. Seven of the ten animals having this shunt had a normal postoperative course, without exhibiting the usual signs of occlusion of the superior vena cava, and with patency demonstrated by angiography or necropsy (Fig. 6). One animal had a small nonoccluding thrombus at the site of the anastomosis. Three animals developed complete superior vena caval thrombosis postoperatively and were killed. In each some technical explanation could be furnished. Thus one animal had the shunt made after removing a great deal of thrombus, which had formed following a previous partial ligation. This thrombus appeared to be of several days' standing. There was a massive pleural effusion, and the intima was seen to be rough in the area from which it was removed. Another had evidently recovered from a partial thrombosis of the superior vena cava, making it necessary to do the anastomosis in an area of the superior vena cava that was narrowed and

thickened. The third postoperative thrombosis occurred in an anastomosis which required an additional suture for bleeding and some twisting had occurred.

Of unusual interest was the following experiment, in which a thrombus weighing 10 Gm. was removed from the superior vena cava. Thrombosis had occurred following partial ligation.

*Animal SVC 2, Male, Weight 11.2 Kilograms.*—Feb. 10, 1948, under nembutal and ether anesthesia the thorax was opened in the usual manner. A braided silk ligature was loosely tied around the superior vena cava just below the azygos vein. This raised the jugular venous pressure from 10 cm. to 17.5 cm. of water.

Feb. 16, 1948, the jugular venous pressure had risen to 28.0 cm., the animal was listless and had edema of the neck and shoulders.

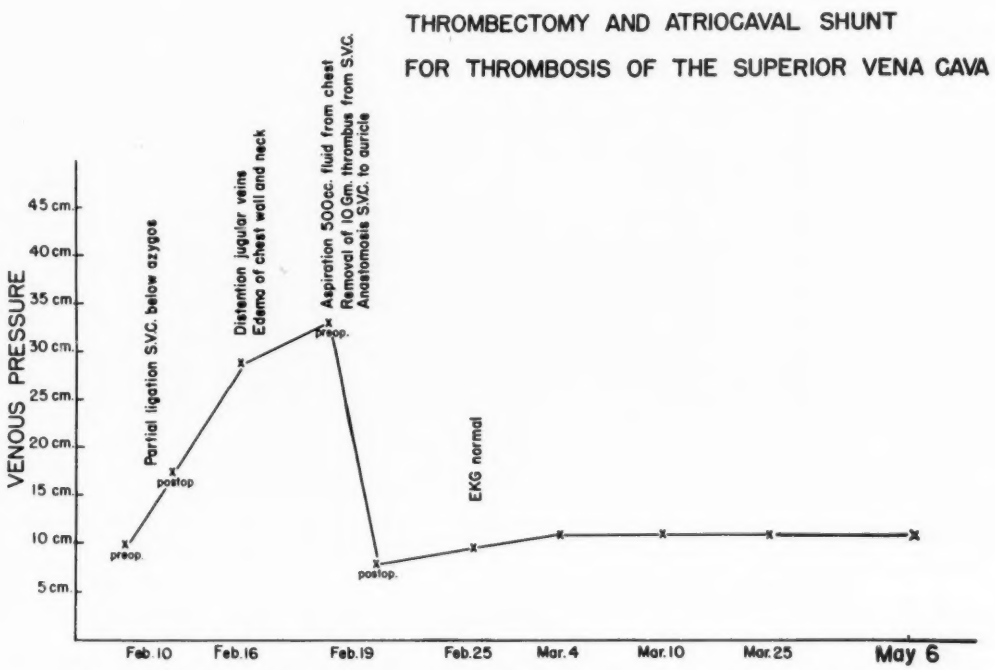


Fig. 7.—Graph showing the venous pressures in Animal SVC 2. Note the rise in jugular venous pressure after partial ligation and thrombosis of the superior vena cava. After removal of the thrombus and establishment of an atriocaval shunt, the venous pressure remained normal.

Feb. 19, 1948. The jugular veins were very distended and the edema had increased. Both sides of the thorax showed signs of pleural effusion. The animal was gasping and appeared moribund. The jugular venous pressure was 33 cm. Under nembutal and ether anesthesia, the thorax was opened and 500 c.c. of serous fluid were aspirated from the pleural space. The superior vena cava was found to be thrombosed for a distance of 3½ cm. It was freed from the surrounding tissues up to the innominate veins, a rubber-covered clamp was applied at this level, and two special clamps were placed just above the azygos. A short incision was then made in the lower end of the superior vena cava and through this opening 10 Gm. of soft thrombus were removed. Clots extending into the innominate veins were washed down by partially releasing the clamp. The lumen was irrigated with saline solution and 1 c.c. of heparin was instilled. The superior vena cava was then transected and the upper divided end was anastomosed to the auricular appendage with an everting suture. The fol-

lowing day the animal was up and about the cage; he made a rapid and uncomplicated recovery. Postoperatively, the jugular venous pressure was 8 cm., and it has continued to remain within the normal limits (Fig. 7). On Feb. 26, 1948, an electrocardiogram was normal, and on March 11, 1948, angiograms demonstrated an open anastomosis (Fig. 8).

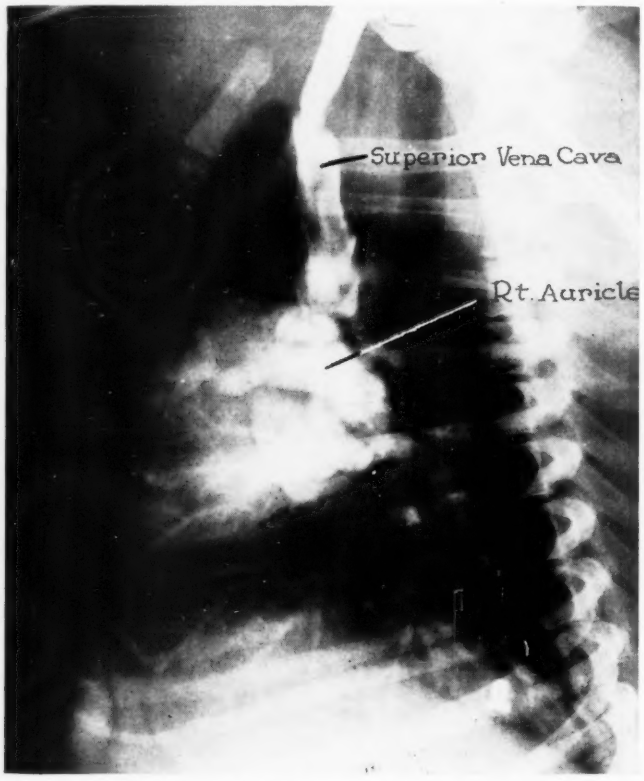


Fig. 8.—Diodrast angiogram of animal SVC 2 three weeks after removal of a thrombus weighing 10 Gm. from the superior vena cava to the right auricle. The dye flows freely into the auricle, without obstruction, although some narrowing can be seen at the site of the anastomosis.

## DISCUSSION

These experiments clearly indicate that venous shunts to the atrium are technically possible, and that if the caliber of the shunt is large it will remain patent in most instances. The atrioazygos shunts demonstrate that some degree of fibrosis must be expected at the site of the anastomosis, and this may be great enough to obliterate a small shunt. The fibrosis is perhaps due to the muscularity of the auricular wall, for it was not seen in the azygos vein to superior vena cava shunts. Some narrowing occurred in the atriocaval shunts, but it was insufficient to produce signs of obstruction in the seven animals which did not develop postoperative thrombosis. Three of these animals have been followed for more than a year. The possibility of arrhythmia was considered, but none was found in those animals which had postoperative electrocardiograms. The fact that our experiments have given some highly successful results without the postoperative use of anticoagulants suggests that similar procedures might be employed in man, under appropriate circumstances.

The level of occlusion of the superior vena cava obviously would determine the type of vascular shunt required. In Fischer's[1] 166 collected cases the obstruction occurred above the junction of the azygos vein in 14, included the azygos in 121, and was below the level of the azygos in 31. This last group would seem to be the most amenable to a shunting procedure, either by utilizing a dilated azygos vein, or by a direct anastomosis of the upper end of the superior vena cava, to the right auricle. When the occlusion involves a long segment of superior vena cava it is conceivable that a dilated innominate or subclavian vein might be used as a shunt to the auricle, or even that a vein graft might prove feasible.

## CONCLUSIONS

Venous obstruction due to experimental occlusion of the superior vena cava at different levels can be successfully relieved by the following types of shunts: azygos vein to superior vena cava, azygos vein to atrium, and superior vena cava to atrium. Shunts from the azygos vein to the atrium in the dog are frequently unsatisfactory due to fibrosis at the line of suture. This disadvantage might be overcome if the vein were larger. Experimentally, anastomoses between the superior vena cava and the auricle are technically possible and they remain patent and functional in most cases. Such vascular anastomoses to the heart might be applied to man under suitable circumstances.

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# THE COEXISTENCE OF INTRA-ABDOMINAL LESIONS IN PATIENTS WITH EPIGASTRIC HERNIA

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IN PATIENTS with epigastric hernia, it should be emphasized that pain in the upper abdomen frequently is caused by a coexisting visceral lesion and not by the hernia. During the period from 1926 to 1948, seventy-six patients with epigastric hernia were admitted to the Portland Veterans' Hospital. In this series, twenty-four patients came to the hospital not because of the hernia but because of upper abdominal pain. The examining physician attributed this pain to the hernia; however, a subsequent study or operation showed the painful lesion to be intra-abdominal, such as an acute duodenal ulcer or an acute cholecystitis.

De Garengeot, in 1743, first described the marked gastrointestinal symptoms associated with this hernia. The coexistence of visceral lesions has been substantiated through the years notably by Alessandrini, Poisson, Mandl, and Monteiro in foreign countries, and by Moschcowitz, Hall, Pemberton, Luke, and Peters in this country.

Epigastric hernia is, by definition, a protrusion of abdominal viscera through an opening in the linea alba at any point between the umbilicus and xiphoid process of the sternum. This implies the presence of a sac, as demonstrated in thirty-six of our forty-eight patients operated upon. A small properitoneal fat tab or portion of the falciform ligament of the liver may wedge through one of the interdigitations which marks the white line of fusion of the external and internal oblique and transverse abdominal muscles. This small fat tab may persist as an interstitial type of hernia in the potential space bounded by the transversalis fascia and peritoneum below and the linea alba above (Pollock). An entering wedge of the round ligament of the liver may form a portion of the wall of a true sac much after the fashion of a sliding hernia. Omentum commonly enters the sac as it did in eleven of our patients to become adherent to the sac wall. The presence of bowel in the hernia or of strangulation is a rarity.

The incidence of epigastric hernia as stated by various authors, such as Watson, Luke, and Peters, is between 1 and 3 per cent of all hernias.

It is probably a much more common lesion for two very good reasons: It is usually symptomless (Gilbride), as in twenty-one patients in our series; and, because it is small, may not be recognized unless the patient is examined while straining or bending backward.

When a patient enters the hospital complaining of pain in the region of an epigastric hernia, it must be determined whether or not the hernia is the cause of this pain. The symptoms produced by the hernia are twofold: The sharp finger-point lancinating pain on straining, well localized to the hernial ring where the filaments of the lower intercostal nerves are impinged; and the deep,

Received for publication, July 16, 1948.

TABLE I

| CASE | INITIALS AND NO. | AGE (YR.) | DURATION | PAIN, LOCAL OR VISCERAL | ENTERED HOSPITAL BECAUSE OF | PRE-OPERATIVE WORK-UP | ASSOCIATED HERNIA | SAC | CONTENTS | ABDOMEN EXPLORED | VISCERAL LESION | OPERATION | RESULT |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1 | F. P. 45347 | 49 | 3 yr. | None | Severe cardiac, repeated admissions | Diverticuli of colon | Bilat. ing. hernia | – | – | – | – | None | Symptomless |
| 2 | H. P. 41282 | 43 | 2 mo. | Local and visceral | Severe upper abdominal pain, vomiting, melena | Angulated 2nd portion of duodenum | None | Yes | Empty | Yes | No visceral path. | Appendectomy and repair | Died 3 yr. later, bronchiogenic Ca., metastases 5 mo. after L. pneumonectomy |
| 3 | G. K. 38122 | 49 | 2 yr. | None | For hernia repair | Neg. | Umbilical and recurrent epigastric | No | – | No | – | Repair of both hernias | Uneventful |
| 4 | C. B. 39295 | 56 | 16 yr. | Local | Long-standing epigastric soreness | Neg. | Small peri-umbilical | Yes | Empty | Palpated | None | Repair | Uneventful |
| 5 | G. S. 40672 | 43 | ? | Visceral | Ulcer type of distress | Neg. G.I. series | None | – | – | – | – | None | Anti acids gave relief |
| 6 | P. H. 40424 | 49 | 3 yr. | Visceral | Sour stomach and vomiting | Penetrating gastric ulcer | None | – | – | – | – | None | Healed on medical management |
| 7 | E. P. 37928 | 65 | ? | None | Kidney colic | Deformed duodenal bulb | None | – | – | – | – | None | Cleared up after cystoscopy |
| 8 | A. W. 38066 | 50 | ? | None | Rt. antral sinusitis | Neg. | None | – | – | – | – | None | Non-symptomatic |
| 9 | J. L. 36301 | 49 | ? | None | Precordial pain and dizziness | Neg. | None | – | – | – | – | None | Non-symptomatic |
| 10 | W. M. 36598 | 44 | 3 yr. | Local | Painful lump in epigastrium | Pruritus ani | None | Yes | Empty | Palpated | Neg. | Repair | Uneventful |
| 11 | F. B. 36738 | 50 | 2 days | Local | Severe localized epigastric pain and mass of 2 days' duration | Deformed duodenum | L. ing. and ventral | Yes | Pus | Yes | Perforated liver abscess | Drainage and repair | No parasites found in stool or pus, OK 3 yr. later |

*(Continued on following pages)*

TABLE I—CONT'D

| CASE | INITIALS AND NO. | AGE (YR.) | DURATION | PAIN, LOCAL OR VISCERAL | ENTERED HOSPITAL BECAUSE OF | PRE-OPERATIVE WORK-UP | ASSOCIATED HERNIA | SAC | CONTENTS | ABDOMEN EXPLORED | VISCERAL LESION | OPERATION | RESULT |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 12 | O. K. 36033 | 57 | ? | None | Nosebleed in hypertensive | Neg. | Rt. ing. | – | – | – | – | None | Cardiac death 8 mo. later |
| 13 | L. E. 34733 | 48 | ? | None | Hypertensive cardiorenal disease | Myocardial insufficiency | None | – | – | – | – | None | Died of gastric hemorrhage 2 yr. later |
| 14 | E. K. 34402 | 49 | 22 yr. | Local | Pain on straining in epigastrium | Neg. | None | Yes | Empty | No | – | Repair | Uneventful |
| 15 | A. S. 34652 | 48 | 1 yr. | Local | Pain locally | Neg. | None | Yes | Empty | No | – | Repair | Mucous colitis and hypochlorhydria found |
| 16 | A. W. 33412 | 64 | 6 mo. | Visceral | Epigastric pain with nausea and vomiting | Achlorhydria, chronic gastritis, small pylorus | None | – | – | – | – | None | 18 yr. before improved on medical management |
| 17 | C. B. 39295 | 56 | 16 yr. | Local and visceral | Painful epigastric mass | Narrow antrum | Multiple epigastric hernias | Yes | Empty | No | – | Repair | Improved |
| 18 | J. W. 57139 | 60 | ? | Visceral | Severe, burning epigastric pain | Nonfilling of G. B. | None | Yes | Omentum | Yes | None found | Repair | Large ventral and hiatal hernia 10 yr. later repaired; good result |
| 19 | D. F. 20801 | 33 | 2 yr. | Local | Painful swelling in epigastrium | Lumbar arthritis | Umbilical | Yes | Empty | No | – | Repair | Suffering from rheumatism 8 yr. later |
| 20 | W. P. 22560 | 63 | 3 yr. | Local | Nausea after meals, pain on straining | Neg. | Rt. ing. | Yes | Empty | No | – | Repair | Cancer of lip 6 yr. later, abd. negative |
| 21 | R. C. 49294 | 43 | 3 yr. | Local | Severe local pain and mass 5 days | Neg. | Rt. ing. | Yes | Round ligament of liver | No | – | Repair of both hernias | Passed a urinary calculus 16 yr. later, no other symptoms |
| 22 | S. A. 15023 | 43 | ? | None | Perirectal abscess | Neg. | None | Yes | Omentum | No | – | Repair | Penetrating gastric ulcer 8 yr. later with tarry stools; diet controlled |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 23 | N. D. 27733 | 43 | 3 mo. | Visceral | Burning epigastric pain, 6 years' duration | Neg. | Bilat. dilated ing. rings | Yes | Empty | Yes | Active duodenal ulcer | Repair and gastroenterostomy | 6 gastric hemorrhages 2 yr. later, 6 yr. later OK |
| 24 | W. K. 21558 | 53 | ? | Local and visceral | Painful hydrocele | Neg. | None | Yes | Empty | Yes | Stellate scar on prox. duodenum | Plication ulcer, repair, and McBurney appendectomy | Uneventful |
| 25 | C. M. 22738 | 46 | 1 yr. | Local on straining | Bleeding hemorrhoids | Neg. | None | Yes | Empty | No | - | Repair | Uneventful |
| 26 | V. B. 23288 | 38 | 3 yr. | Local on straining | Painful epigastric mass | Neg. | Umbilical | No | - | Yes | G. B. adherent to mesocolon: appendix adherent | Appendectomy and repair of hernias, G.B. freed | Uneventful |
| 27 | F. C. 6369 | 31 | 10 yr. | None | Pain rt. groin, persistent cough | Neg. | Rt. ing. | Yes | Omentum | No | - | Repair of hernias | Uneventful |
| 28 | G. M. 59962 | 52 | 5 yr. | Local | Pain on straining | Hiatal hernia | Hiatal hernia | Yes | Empty | No | - | Repair | Uneventful |
| 29 | J. R. 29503 | 46 | 20 yr. | Local | Mass in epigastrium | Neg. | None | Yes | Empty | No | - | Repair | Uneventful |
| 30 | R. P. 33406 | 50 | ? | Visceral | Nervous, upset stomach | Free Hcl 76 units | Bilat. ing. and rectus femoris | Yes | Empty | Yes | Duodenal ulcer | Repair | Complete relief on medical management |
| 31 | B. B. 28663 | 50 | 35 yr. | Local | Gnawing, enlarging epigastric mass | Neg. | None | Yes | Empty | No | - | Repair | Uneventful |

*(Continued on following pages)*

TABLE 1—CONT'D

| CASE | INITIALS AND NO. | AGE (YR.) | DURATION | PAIN, LOCAL OR VISCERAL | ENTERED HOSPITAL BECAUSE OF | PREOPERATIVE WORK-UP | ASSOCIATED HERNIA | SAC | CONTENTS | ABDOMEN EXPLORED | VISCERAL LESION | OPERATION | RESULT |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 32 | A. N. 45938 | 66 | 12 yr. | Local and visceral | Severe epigastric burning of 10 days | Hyperacidity, poor filling of duodenal cap and coffee-ground vomitus 10 days before | Umbilical | - | - | - | - | None | Ulcer regime, completely controlled symptoms |
| 33 | W. T. 45730 | 53 | 28 yr. | Local | Unable to lift because of epigastric pain | Neg. | None | Yes | Omentum | No | - | Repair | Uneventful |
| 34 | C. A. 45650 | 56 | ? | - | Fractured cartilage, Rt. rib cage | Neg. G.I. series | None | - | - | - | - | None | Asymptomatic |
| 35 | M. K. 45476 | 27 | 4 yr. | Local and visceral | Local soreness and enlargement of mass | Gastric hemorrhage 3 yr. prior, present G.I. series neg. | None | Yes | Empty | No | - | Repair | Uneventful |
| 36 | C. R. 36003 | 58 | 10 yr. | Local and visceral | Pain after meals, 2 yr. | Deformed duodenal cap | None | - | - | - | - | None | Controlled on ulcer treatment, urinary calculus 3 yr. later |
| 37 | S. S. 44966 | 54 | 10 yr. | Local and visceral | Severe epigastric burning, 10 days | Hyperacidity, poor cap visualization, coffee-ground vomitus 10 yr. before | Umbilical | - | - | - | - | None | Ulcer management completely controlled |
| 38 | R. R. 37715 | 46 | 4 mo. | Visceral | Severe pain, rt. upper abdomen | Neg. | None | No | - | Yes | Perf. duodenal ulcer against G. B. | Closure, perforation, and repair | Improved |
| 39 | R. P. 33406 | 50 | 1 yr. | Visceral | Nervous chronic upset stomach | Neg. G.I. series | Bilat. ing. | Yes | Empty | No | - | Repair | Anti acids relieved stomach pains |
| 40 | B. D. 30952 | 44 | 5 yr. | None | Recurrent epigastric hernia | Neg. | None | Yes | Omentum | No | - | Repair | Uneventful |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 41 | O. J. 30159 | 49 | 5 yr. | Visceral | Jaundice, weight loss, epigastric pain | Neg. | None | Yes | Omentum | Yes | Cholecystitis; fibrosed appendix | Cholecystectomy; appendectomy; repair | Uneventful achlorhydria and atrophic gastritis 1 yr. later |
| 42 | B. C. 46457 | 58 | 23 yr. | Visceral | Hepatitis | Neg. | None | – | – | None | – | None | Responded to medical treatment |
| 43 | W. P. 57105 | 30 | 6 mo. | Visceral | Pain RLQ, nausea and vomiting | Lesion of rt. apex | None | Yes | Empty | No | – | Appendectomy and repair | Uneventful |
| 44 | Z. J. 55893 | 67 | 3 mo. | Local | Local epigastric pain | Bronchitis and emphysema | None | No | – | No | – | Repair | Uneventful |
| 45 | E. B. 55545 | 51 | 12 yr. | Visceral | Epigastric pain | Penetrating gastric ulcer | None | No | – | Yes | Penetrating ulcers of duodenum and stomach | Subtotal gastric resec. and repair | Uneventful |
| 46 | W. E. 40931 | 49 | ? | None | Fatigue | Scoliosis and arthritis | None | – | – | – | – | None | Asymptomatic |
| 47 | B. B. 28663 | 50 | 35 yr. | Local | Gnawing epigastric pain | Neg. | None | Yes | Empty | No | – | Repair | Uneventful |
| 48 | J. R. 29503 | 46 | 20 yr. | None | For repair | Old compression fracture, D.10 | None | No | – | No | – | Repair | Uneventful |
| 49 | F. F. 43050 | 49 | ? | None | Cough | Myocardial degeneration | Bilat. ing. and umbilical | – | – | – | – | None | No abdominal symptoms 5 yr. later |
| 50 | T. S. 27665 | 49 | 11 yr. | Local | Constant epigastric pain | Neg. | None | No | – | No | – | Repair | Gastroenteritis 7 yr. later |
| 51 | J. K. 26946 | 43 | 3 yr. | Local | Pain on strain | Neg. | None | Yes | Empty | No | – | Repair | Uneventful |
| 52 | M. P. 26478 | 61 | 7 yr. | Local | Fracture of left tibia | Neg. | None | – | – | – | – | None | Asymptomatic |

*(Continued on following pages)*

TABLE I—CONT'D

| CASE | INITIALS AND NO. | AGE (YR.) | DURATION | PAIN, LOCAL OR VISCERAL | ENTERED HOSPITAL BECAUSE OF | PRE-OPERATIVE WORK-UP | ASSOCIATED HERNIA | SAC | CONTENTS | ABDOMEN EXPLORED | VISCERAL LESION | OPERATION | RESULT |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 53 | F. F. 29826 | 45 | ? | Visceral | Burning epigastric pain for 9 yr. | Duodenal ulcer | None | - | - | - | - | None | Continued to have pain on medical management |
| 54 | G. V. 32309 | 53 | 1 yr. | Visceral | Epigastric distress and gas | Neg. G.I. series | None | - | - | - | - | None | Not treated |
| 55 | C. H. 32273 | 54 | ? | None | Fracture of pelvis and femur | Neg. | None | - | - | - | - | None | Asymptomatic |
| 56 | B. D. 30952 | 44 | 3 yr. | None | Recurrence at 4 yr. | Emphysema | None | Yes | Omentum | No | - | Repair | Uneventful |
| 57 | E. G. 37362 | 81 | 7 yr. | None | Dyspnea and cough | Bronchial asthma and emphysema | Rt. ing. | - | - | - | - | None | Asymptomatic |
| 58 | O. S. 24381 | 38 | 8 day | Local | Painful lump in epigastrium | Nasal polypi | None | Yes | Omentum | Yes | None | Repair | Good |
| 59 | L. W. 24965 | 56 | 20 yr. | Local | Tender epigastric mass on strain | Neg. | None | Yes | Empty | No | - | Repair | Uneventful, asymptomatic 2 yr. later |
| 60 | J. S. 12996 | 37 | 60 days | Local | Pain in epigastrium after meals | Neg. | Bilat. ing. | Yes | Empty | No | - | Repair | Uneventful asymptomatic 1 yr. later |
| 61 | E. W. 24989 | 39 | ? | None | Bronchitis, influenza | Neg. G.I. series, bronchial asthma | Rt. femoral | Yes | Omentum | No | - | Repair | Uneventful |
| 62 | G. W. 18049 | 37 | 1 yr. | Local | Pain on strain | Manic depressive psychosis | None | No | - | Yes | Tense G. B. | Repair and appendectomy | Uneventful |
| 63 | G. O. 18457 | 42 | ? | Local | Pus in urine and dribbling | Diabetes and syphilis | None | - | - | - | - | None | Asymptomatic |
| 64 | W. K. 19282 | 34 | 15 mo. | Local | Pain in epigastric mass | Neg. | None | No | - | Yes | None | Repair | Uneventful |
| 65 | T. I. 20289 | 45 | 3 yr. | Visceral | Distress upper abdomen | Gastroduodenitis | None | - | - | - | - | None | Refused surgery |

| | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 66 | A. M. 55108 | 52 | 2 yr. | Visceral and local | Epigastric soreness and eructations after meals | Neg. | None | Yes | Omentum and round ligament | No | – | Repair | Uneventful, 6 mo. later no recurrence |
| 67 | J. S. 54910 | 63 | 20 yr. | Local | Arthritis, spine | Neg. | None | – | – | – | – | None | Asymptomatic |
| 68 | N. S. | 52 | 5 yr. | Local | Pain in epigastrium | Diverticulum of 3rd portion of duodenum | None | Yes | | | | | |
| 69 | H. D. 46022 | 51 | ? | ? | Dyspnea | Neg. | Rt. ing. | – | – | – | – | | Operation prior to 1926—no recurrence—1946 |
| 70 | D. W. 58277 | 23 | 1 yr. | Local | Pain after meals | Neg. G.I. series | Bilat. ing. | – | – | – | – | – | No repair of epigastric hernia |
| 71 | M. H. 59127 | 27 | 3 yr. | Local | Pain on strain, dull and local | Neg. | None | Sac | Omentum | No | – | Repair | Uneventful |
| 72 | W. J. 59175 | 39 | 1 yr. | Visceral | Postprandial epigastric pain | Periesophageal hernia and scarred duodenal bulb | Rt. ing. | – | – | – | – | None | Complete relief on medical management |
| 73 | J. S. 47426 | 46 | 4 mo. | None | Furunculosis | Neg. | Rt. ing., rt. ventral, rt. femoral | No | – | No | – | Repair of multiple hernias | Uneventful |
| 74 | C. V. 34259 | 44 | ? | None | For evaluation of compensation claim | Pulm. tbc., inactive | None | ? | ? | ? | ? | Repair | Recurrence 4 yr. later |
| 75 | V. N. 51718 | 26 | 1 yr. | Local | Severe stinging epigastric pain | Neg. | None | No | – | No | – | Repair | Acute duodenal ulcer responded to therapy 5 mo. later |
| 76 | J. A. | 60 | ? | ? | ? | Neg. | Bilat. ing. | ? | ? | No | – | Repair | Narrow pylorus, eventration of L. diaphragm 4 yr. later |

boring, aching pain aggravated by eating or bending backward, produced by drag on the greater omentum, greater curvature of the stomach, or falciform ligament of the liver. Pain is relieved on reduction of the mass, or on lying down, aggravated by straining, bending backward, or eating a full meal. Thirty of our patients entered the hospital with these symptoms.

Those patients with vague roentgenologic findings and poor response to therapy for suspected biliary or upper gastrointestinal disease should have a careful, detailed examination for epigastric hernia (Moscheowitz). The epigastric mass may remain hidden beneath the linea alba (Napalkow).

Of even greater importance are those patients with epigastric hernia and ulcer syndrome or vague visceral complaints. In these patients, the hernia may be discovered incidentally, or the periodic pain may be relieved by food or alkali. These patients must have adequate roentgenologic examination before undergoing surgery for the hernia. A trial period of medical management may entirely relieve their symptoms. By this regime, surgery was deemed unnecessary in five cases. If these patients come to surgery, the abdomen should be explored at the time of repair. This decision should not be altered by negative roentgenograms. Thus, in six patients with negative preoperative diagnostic studies, specific lesions were found on exploration. These were a stellate scar of the duodenum, a chronic cholecystitis, a gall bladder adherent to the mesocolon, two acute duodenal ulcers, and one duodenal ulcer perforated against the gall bladder.

Occasionally, repair of the hernia was effected without recognizing the intra-abdominal lesion. Because of the persistence of pain following surgery, a more careful and detailed study of these patients was made; the true nature of their complaints was then readily revealed. One patient (Case 75, see Table I), returned to the hospital six months after operation with persistent epigastric distress. An acute duodenal ulcer was demonstrated on the gastrointestinal series and medical management completely controlled the pain. Another patient (Case 18) was found to have a peri-esophageal hernia ten years following a negative exploration and epigastric hernial repair. Following the operation for hiatal hernia, he experienced immediate and complete symptomatic relief.

There were four other patients operated upon who returned because of severe and persistent upper abdominal pain. One patient (Case 41) developed an atrophic gastritis one year after surgery; another (Case 23) had a gastric hemorrhage two years later; one patient (Case 76) revealed an eventration of the left diaphragm with a narrowed pyloric antrum four years following surgery, and another (Case 22) bled from a penetrating gastric ulcer eight years after repair of the hernia. One patient, without previous surgery, died two years later of a gastric hemorrhage.

## CONCLUSIONS

1. In a series of seventy-six patients admitted to the Portland Veterans' Hospital for epigastric hernia, twenty-four showed coexisting upper abdominal pathology.

2. The need for careful preoperative roentgenograms of stomach, gall bladder, and colon is essential.

3. Exploration of the abdomen in patients with epigastric hernia and vague visceral symptoms is emphasized.

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# A NEW, SAFE, SIMPLE APPARATUS FOR OBTAINING NEGATIVE SUCTION FOR THE PATIENT WITH AN INDWELLING GASTROINTESTINAL TUBE

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SINCE the innovation of suction applied to inlying tubes in the gastrointestinal canal,[1] a widespread utilization of this principle has resulted with a constantly expanding list of indications for its use. Three-bottle nasal suction probably has enjoyed the greatest popularity in spite of the introduction periodically of new machines designed to accomplish the same purpose. Most apparatuses have satisfied many of the necessary requisites for a satisfactory unit. However, the disadvantages have been real. These disadvantages have been related mainly to (1) the inability to regulate the amount of negative pressure and (2) the great number of potential leaks. (In one appliance there are fifty-five potential sources of leak.) Of course, there are other means by which negative suction can be obtained. However, these methods also have disadvantages which negate any potential advantage each may have. The main methods available are wall suction and suction created by an electric motor pump. In each instance the amount of suction can be controlled by means of a break-over manometer. The disadvantages of these methods are obvious. In both types of suction, if constant attention is not paid to the level of the fluid in the catch bottle and it is allowed to become overfilled, or is accidentally knocked over, serious damage may be done. In the first instance the negative suction system of the hospital may be damaged, which, disregarding the inconvenience to the hospital, will result in a considerable expense for the repair of the situation. An electric motor pump similarly is vulnerable to such damage and, in addition, is a constant source of annoyance to the maintenance department.

At the University of Minnesota Hospitals, until recently, three-bottle suction has been preferred to the exclusion of all other mechanical appliances for the patient with an indwelling tube in the gastrointestinal canal. Three-bottle suction has been used because of the advantage of being able to adjust the amount of negative pressure, and the inexpensiveness of the materials used. This latter "advantage" is implied rather than real. In the experience at the University Hospital approximately twenty suction sets are in constant use. In order to keep this many three-bottle sets in operation, the reserve of materials must be sufficient to assemble forty sets. In spite of this, it is probably true that the surgical supply department would have difficulty in assembling twenty-five complete sets which would work satisfactorily. The expense of maintenance far overshadows the initial cost; bottles are constantly being broken (at the University of Minnesota two to three per day at 35 cents each), stoppers wear

Received for publication, July 11, 1948.

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out and are inadequate and a source of leakage; in addition, there is the added expense associated with the cleaning of the bottles, stoppers, and tubing. In investigating the expenditures at this hospital, for only the replacement of bottles and the repair and replacement of stoppers excluding the item of tubing and the cleaning of the equipment, it was found that the cost to this institution in 1945 was $601; in 1946, $734 (for the first three months of this year the expenditure was $336); and for the first ten months of 1947, $655. This is a continual expense. Certainly the importance of this item cannot be underestimated.

Because of the inconvenience of transporting and storing three-bottle sets, the difficulty of maintenance and assemblage of a satisfactory set without leaks, the inherent danger to the patient when assembled incorrectly, and the expense involved, one of us (J.A.P.) devised an apparatus to be described. This instrument has proved so satisfactory and simple that it is gradually supplanting the three-bottle sets in this institution.

## DESCRIPTION OF THE APPARATUS

The apparatus consists mainly of a hollow tank with a crown top and inverted bottom. This steel cylinder measures 15¾ inches in diameter by 24 inches in height. It is made of 14 gauge steel of welded construction throughout. It is supported by four Faultless casters with a load rating of 200 pounds. The tank itself weighs approximately 40 pounds. Secured in the crown top is a suitable handle for moving the piece, a negative pressure gauge graduated in inches of mercury, and a single orifice needle valve. On the side opposite from the handle and recessed into the cylinder is a hand pump. A machine-surfaced flange fastens the complete pump into the crown by means of seven machine screws. The parts of the pump are chosen with extreme care in order to embody long life and to insure a freedom from maintenance and repair. The pump cylinder is a heavy walled brass tube. The check valve at the bottom of the pump cylinder is made of oilproof neoprene. The pump leather has been specially designed. It is fastened in such a manner to the bottom of the pump rod, that it never can become loose or unscrewed. The complete pump assembly can be readily removed in case attention is necessary. In the event of an inadvertent overflow of the aspirated material into the tank, because of a failure to empty the catch bottle, the removal of the pump assembly affords an adequate 2 inch opening for washing and emptying the interior of the cylinder. Between the handle and the pump in the center of the crown is the negative pressure gauge. In juxtaposition to this gauge is a conduit to which is connected the rubber tubing which connects to the catch bottle and then to the patient. All fittings are constructed of satin chrome plated brass. The steel tank is bonderized (rust proofed) inside and out. The synthetic lacquer finish (hammered aluminum) is baked for reasons of durability (Fig. 1).

## PRINCIPLES INVOLVED

With the needle valve closed about twenty-five to thirty light, slow strokes of the pump handle will evacuate the steel tank and the gauge will then show approximately five inches of negative mercury pressure. After the nasal tube

is connected to the catch bottle, the needle valve is opened. The drainage bottle is immediately evacuated as well as the nasal tube. The suction to the drainage bottle and thence to the patient is never interrupted, even while evacuating the tank. Considering the steel tank as a reservoir of negative pressure, the same having been built up by twenty to thirty strokes of the pump, this quantity is sufficient to drain approximately four gallons of liquid and/or air from the patient. Consequently, it is necessary to change the gallon drainage bottle periodically. Ordinarily, this lasts the average patient twenty-four to thirty hours.

Fig. 1.—A photograph of the suction apparatus in use. A model now is available which is lower and may be placed under the bed thereby allowing additional room and increasing the accessibility to the patient.

## ADVANTAGES

*To the Patient.*—The keynote of the advantage to the patient is safety. This device is foolproof. Because of the construction of the machine, it is impossible to create a positive pressure, therefore avoiding the danger of insufflating the stomach with air. In addition, because of the absence of water in the

suction system, the danger of flooding the patient is obviated also. The amount of suction can be varied and in no instance after the initial pressure is established can the negative pressure be excessive, as the supply of negative suction decreases as the apparatus functions.

*To the Nurses.*—The bedside care of the appliance is negligible. The control pressure in the tank (usually five inches of negative pressure) is easily established by thirty excursions of the pump incorporated in the apparatus; the replenishment of negative pressure is equally simple. Because of the simplicity of the machine, valuable time is saved with regard to nursing care. The time saved may be used to advantage in the care of the patient involved or of other patients. The tank occupies minimal space, and in this manner allows easy accessibility to the patient.

*To the Institution.*—The machine is easily portable and occupies a minimum of space. It may serve a number of patients at one time utilizing individual drainage bottles. The expense of maintenance, replacement, washing, and the storage of bottles and tube as is necessary with three-bottle sets is practically nonexistent. The saving in the cost of maintenance, nursing care, and repairs with this appliance far outweighs any advantages of the three-bottle suction sets or electrical contrivances.

*Mechanical Advantages.*—The simplicity of the apparatus is apparent. The maintenance of pieces is limited to three as compared to fifty-five in certain other machines. Regarding the matter of replacements, standard parts are used which may be obtained at any automobile accessory shop, and can be inserted easily and rapidly at a reasonable expense. If the collection bottle interposed between the patient and the tank overflows into the tank, by simply removing the pump assembly one gains access to the interior of the cylinder. Through this 2 inch aperture the inside of the cylinder may be irrigated, cleansed, and emptied. The freedom from electrical controls makes the apparatus applicable in either the hospital, home, or office. The potential sources of leak are reduced to a minimum.

## SUMMARY

A new apparatus for obtaining negative suction, applicable to the patient with an indwelling gastrointestinal tube, has been described. Certain advantages of this device have been pointed out as well as the disadvantages of other machines.

The outstanding features of this apparatus relate themselves to safety, simplicity of operation, the marked reduction of the potential sources of leakage, the lack of expense incident to maintenance and replacement of parts, freedom from electrical connections or sensitive controls, portability, and adaptability to the hospital, home, or office.

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# A MODIFIED SYNTHETIC PREDIGESTED ALIMENT FOR PROTRACTED JEJUNOSTOMY FEEDING

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IN A historical review of the literature from 1858 to 1944, Rosenak and Hollander[5] discussed various feeding mixtures which have been employed for jejunal alimentation, as well as the numerous clinical conditions in which this surgical jejunostomy was used. Since 1858, when the first instance of jejunal alimentation was recorded, the character of the feeding material used for this purpose has undergone change from natural foodstuffs, such as the meat–eggs–meat-broth diet of Busch,[2] to a synthetic mixture of the nutritional essentials in relatively simple form. In general, the feeding of these mixtures frequently resulted in nausea, vomiting, cramps, and diarrhea. These disturbances were discussed by Hollander, Rosenak, and Colp in 1945[4] in a report describing a preparation which was designed to avoid such complications. When not referable to the postoperative state, these untoward reactions can be attributed to one or more of the following factors: (1) particular components of the feeding mixture, for example, high fat content, glutamic acid, irritant substances, etc.; (2) a high rate of administration, greater than the patient's tolerance; (3) excessive osmotic pressure; (4) extremes of pH. The success of the aliment devised in this laboratory was ascribed particularly to the control of these factors.

However, this nutritive preparation was originally designed for short-term feeding only, either pre- or postoperatively. Since its introduction at this hospital, the demand for the aliment has increased extensively; it has been employed in a variety of conditions in young children as well as adults. It has been found particularly useful in conditions where it was required to supply all or most of the patient's nutrition for prolonged periods. Among these were included cases of anorexia nervosa requiring tube feeding by mouth, cicatricial stenosis of the esophagus with gastrostomy, and jejunostomy for gastrointestinal obstruction. However, since the original aliment contained only the major vitamins, it was considered inadequate for such prolonged feeding. In addition, some difficulty was encountered in supplying the material to the patient, because of the large quantities of the aliment which had to be prepared fresh and sterilized every day. When a part of

Presented at a meeting of the Thirty-Third Annual Clinical Congress of the American College of Surgeons, New York, N. Y., Sept. 10, 1947.

This paper reports research undertaken in cooperation with the Quartermaster Food and Container Institute for the Armed Forces, and has been assigned a number *185* in the series of papers approved for publication. The views or conclusions contained in this report are those of the authors. They are not to be construed as necessarily reflecting the views or indorsement of the Department of the Army.

The authors are indebted to Mrs. Anita Bamberger, R.N., for technical assistance in the course of this work.

Received for publication, July 19, 1948.

the day's supply was lost by contamination or by accident, or there was an unexpected call for aliment for a new patient, either a second batch had to be prepared immediately—a time-consuming procedure—or a patient was deprived of his feeding for that day. In order to obviate such difficulties and to insure that the aliment would be nutritionally adequate for long-term administration, the formula has been modified in some of its details. Also, a simplified preparation technique was devised, whereby a large supply could be made at one time and kept for two or more weeks without significant loss of vitamin potency or increase in bacterial count.

The modified preparation has been formulated so that 2,000 Calories (2 liters) will supply the following:*

(1) All the essential amino acids, in quantities sufficient for growth and repair of tissue.

(2) Fat to supply the essential fatty acids, in quantities great enough to furnish some of the calories in a noncarbohydrate form (and thus avoid excessive osmotic pressure) and yet not induce intestinal motor disturbances.

(3) Carbohydrate sufficient to replace depleted glycogen stores, spare protein, make up the residual caloric requirement, and yet avoid fermentation and diarrhea.

(4) An adequate supply of the minor vitamins in addition to the six major vitamins for which recommendations were made by the National Research Council Committee on Foods and Nutrition in 1945.

(5) All essential minerals, in amounts suggested by the National Research Council recommendations and a survey of 150 American dietaries reported by Sherman in 1941,[7] with sufficient NaCl to restore and maintain adequate water and electrolyte balance.

(6) Water, in quantity designed to maintain the osmotic pressure within reasonable limits, and to dilute the aliment so that it flows readily through the drip apparatus.

If 2,000 Calories be adopted as a basal requirement, 2 liters of the aliment will supply the various nutritional components in amounts necessary for these minimum specifications (Table I). The aliment is prepared in 500 Calorie units, of 500 c.c. total volume each, so that the caloric density is 1 Calorie per milliliter. Twenty-five per cent of the calories are derived from protein, 15 per cent from fat, and 60 per cent from carbohydrate; each unit contains at least one-half of the daily vitamin requirement recommended by the National Research Council in 1945. The vitamin D content is sufficient for adults but must be supplemented independently for children. Hence, a patient being fed 2 liters of this mixture per day would receive twice the NRC recommendation of vitamins, and a patient receiving only 1 liter would still be assured of the minimum.

*For a detailed analysis of the considerations which led to the quantitative composition of this preparation, see the paper by Hollander, Rosenak, and Colp.[4]

TABLE I. COMPOSITION OF 2,000 CALORIES (2 LITERS) OF ALIMENT "MSH 47"

| | | |
|---|---|---|
| Protein (500 Calories) | 125 | Gm. |
| Carbohydrate (1,200 Calories) | 300 | Gm. |
| Fat (300 Calories) | 33.4 | Gm. |
| Vitamin A | 12,240 | I.U. |
| Vitamin D | 204 | I.U. |
| Mixed tocopherols | 100 | mg. |
| Ascorbic acid | 200 | mg. |
| Thiamine | 10.5 | mg. |
| Riboflavin | 20 | mg. |
| Nicotinamide | 50 | mg. |
| Pyridoxine | 3 | mg. |
| Liver (whole, desiccated) | 20 | Gm. |
| NaCl | 10 | Gm. |
| Potassium | 4.16 | Gm. |
| Phosphorus | 2.10 | Gm. |
| Calcium | 1.48 | Gm. |
| Magnesium | 0.35 | Gm. |
| Iron | 0.03 | Gm. |
| Manganese | 0.008 | Gm. |
| Copper | 0.002 | Gm. |
| Iodine | 0.0003 | Gm. |
| Water | 1,700 | ml. |

The aliment is made up in 3 separate parts, each of which is prepared in bulk but bottled in unit containers for storage. These 3 parts are (1) a dry mixture, containing the protein, carbohydrate, salts, liver, and ascorbic acid, which is sterilized after being packaged (bottle A); (2) an aqueous fat emulsion, containing the fat component and the fat-soluble vitamins including A, D, and E (tube B); and (3) the water-soluble vitamins—vitamin B complex (tube C).

TABLE II. COMPOSITION OF THE DRY MIXTURE

| | | |
|---|---|---|
| No. of package units | 1 | 50 |
| Calories | 500 | 25,000 |
| Lactamin (Gm.) | 37 | 1,850 |
| Dexin (Gm.) | 75 | 3,750 |
| Whole liver substance (Gm.) | 5 | 250 |
| Ascorbic acid (Gm.) | 0.05 | 2.5 |
| Salt mixture, MSH 47 (Gm.) | 5.4 | 270 |
| Total weight of mixture (Gm.) | 122.45 | 6,122.5 |

*The dry mixture (bottle A)* consists of protein hydrolysate (Lactamin*), dextrin-maltose mixture (Dexin†), whole liver substance‡, ascorbic acid powder, and a special salt mixture (MSH 47), in quantities indicated in Table II. This mixture is packaged in units of 500 Calories each, but it is prepared in bulk sufficient to make 50 or 100 such units—in which case the several components are taken in proportionate amounts. The salt mixture is usually prepared in kilogram batches. Its composition, shown in Table III, is such as to yield the quantity of minerals indicated in Table I, if the salt content of the other components of the aliment is also considered. The required amounts of the dry materials are weighed into a large covered container and

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†Burroughs Wellcome & Co., Inc., New York, N. Y.
‡Wilson Laboratories, Division of Wilson & Co., Inc., Chicago, Ill.

TABLE III. COMPOSITION OF SALT MIXTURE (MSH 47) (GRAMS PER KILOGRAM)

| | |
|---|---|
| NaCl | 443 |
| $K_2HPO_4$ | 426 |
| $MgSO_4 \cdot 7H_2O$ | 126 |
| $FeSO_4 \cdot 7H_2O$ | 5 |
| $CuSO_4 \cdot 5H_2O$ | 0.15 |
| KI | 0.02 |

mixed thoroughly. If a suitable mechanical device is not available, the material may be mixed in a can by shaking, rolling, inverting, etc., until it is well blended. One hundred twenty-three grams of the dry mixture are weighed into a clean, dry, quart milk bottle. Each bottle is closed with a dated bottle cap and a tie seal is made with a dated souffle cup. The sealed bottle is then kept in a steam sterilizer (not an autoclave) at atmospheric pressure and 212° F. for 20 minutes. After cooling, these bottles are stored in a refrigerator until needed. Bottles of the dry mixture, with seals unbroken, have been kept for almost a month without any significant increase in bacterial count. Bacterial growth, if observed at the time of actual feeding of the aliment, may be ascribed to contamination during the addition of the fluid components to the dry powder in bottle A.

*The fat-water emulsion (tube B)* has the composition shown in Table IV. Usually one liter is made at a time, and this is divided into units of 17 ml. each—enough to supply the amount of fat-soluble vitamins and fat calories requisite for a single 500 Calorie unit of aliment. The tocopherols and halibut liver oil are added to the corn oil, and the solution is mixed intimately with the acacia in a large mortar. About one-half of the water is then ground with this liquid until the whole is uniformly dispersed, after which the remainder of the water is added slowly with constant grinding. At this stage, the emulsion will separate but it can easily be resuspended by shaking. However, after being put through a hand homogenizer mill it becomes sufficiently stable for all subsequent purposes. The emulsion is measured into test tubes (17 ml. to the tube), capped, and stored in the refrigerator. Assays for vitamin A in four such preparations, with and without mechanical homogenization, revealed no significant loss of potency for four weeks following their preparation.*

TABLE IV. COMPOSITION OF THE FAT-WATER EMULSION*

| | |
|---|---|
| Corn oil | 500 ml. |
| Halibut liver oil (vitamins A and D) | 3.5 ml. |
| Mixed tocopherols (vitamin E) | 1.5 Gm. |
| Acacia | 125 Gm. |
| Water | 500 ml. |

*One package unit of ailment (500 Calories) contains 17 ml. of this emulsion.

*Water-soluble vitamins (tube C)* may be supplied by any suitable commercial preparation. We have found it convenient to use Blexin (I.V.C.)† supplemented with nicotinic acid (75 mg. per 100 ml.). Enough of the water-soluble vitamins for one 500 Calorie unit of the aliment (except for vitamin C,

*These vitamin A assays were kindly performed for us by Dr. Susan Kann, using the method of Bessey and co-workers.[1]

†International Vitamin Division, American Home Products Corp., New York, N. Y.

which is included in bottle A) is contained in 12.5 ml. of this Blexin-nicotinamide mixture. Hence, the latter is prepared in bulk, but stored in small test tubes, each of which contains this volume of the mixture. If it is so desired, particularly for purposes of controlled investigation, the Blexin-nicotinamide mixture and the whole liver substance may be replaced by a capsule containing a mixture of the crystalline vitamins (Table V). When this alternative procedure is employed, a small additional amount of Dexin (6 Gm. per 500 Calorie unit) is added to the dry mixture as an isocaloric substitute for the liver preparation; also since the ascorbic acid is contained in the capsule it is omitted from bottle A. The constituents of the vitamin mixture are blended in a mortar and put in capsules (No. 00). One capsule, containing 0.8 Gm., is used for each 500 Calorie bottle of aliment.

TABLE V. COMPOSITION OF THE CRYSTALLINE VITAMIN MIXTURE (WEIGHTS IN GRAMS FOR 1000 CAPSULES)*

| | |
|---|---|
| Thiamine | 0.9 |
| Ascorbic acid | 50.0 |
| Riboflavin | 1.25 |
| Pyridoxine | 0.75 |
| Nicotinamide | 9.0 |
| Biotin | 0.07 |
| Calcium pantothenate | 5.5 |
| Pteroylglutamic acid | 0.63 |
| p-Aminobenzoic acid | 15.0 |
| Choline | 100.0 |
| Inositol | 500.0 |
| Lactose | 116.9 |
| Total weight | 800.0 |

*One package unit of aliment (500 Calories) contains 0.8 Gm. or 1 capsule of this mixture.

*Preparation of the Fluid Aliment.*—In order to prepare one 500 Calorie unit of the feeding mixture with a caloric concentration of 1 Calorie per milliliter, 425 ml. of warm sterile water are added with aseptic technique to bottle A, containing the dry mixture. The bottle is well shaken until all the dry material is in solution. Then, one unit each of the fat emulsion (tube B) and the water-soluble vitamins (tube C or the contents of one capsule) are added. The mixture is shaken vigorously and poured into the reservoir of the drip or other dispensing apparatus, making certain that the preparation is no colder than room temperature. For convenience in storage in the refrigerator and delivery to the patient's room, the two test tubes (B and C) are taped to the bottle of dry mixture so that they can be handled as a unit.

### PRECAUTIONS IN THE ADMINISTRATION OF THE ALIMENT

1. Since the aliment is an excellent culture medium for bacteria when wet, and the contents of bottle A are hygroscopic, this bottle must be adequately sealed and sterilized after preparation; also, it should be stored in a cool dry place. The aqueous fat emulsion (tube B) must be kept in a refrigerator, in order to prevent oxidation of the fats and the vitamin A. The water-soluble vitamin mixture (tube C) need only be kept cool. All equipment employed for preparation and administration of this material must be cleaned

thoroughly and dried *after each use*. Furthermore, the drip apparatus used for feeding the aliment must be sterilized, and actual administration conducted with regard for the principles of asepsis. When the sterile water, vitamins, and oil emulsion are added with ordinary care (without flaming of bottles), the final mixture may be kept at room temperature for at least four hours without significant bacterial growth.

2. The mixture must never be introduced into the small intestine when cold. Warming the aliment to body temperature *in a water bath* before it is placed in the drip reservoir will obviate this possibility.

3. In order to insure the absence of nausea, vomiting, etc., particular care must be taken to avoid an excessive rate of administration of the aliment. If the feedings are started within the first few days after operation, the initial rate must not exceed 50 ml. per hour.* Thereafter, it may be increased gradually, in accordance with the patient's tolerance, until a maximum of 150 to 200 ml. per hour is reached—usually about one week later. Under no circumstances should the early convalescent patient be given the feedings faster than 200 ml. per hour. In patients with a permanent or semipermanent jejunostomy, however, the rate may be increased gradually to a value considerably greater than this; the limit can be established only by the individual's tolerance.

4. For routine feedings of hospitalized patients, the total daily intake should be at least 2,000 ml., and preferably 2,500 ml. In some cases, the total may be increased still further. Of course, it is necessarily very much less during the first few days after operation. The total daily quantity at the higher levels of intake may be distributed into several feedings of 500 Calories each (500 ml.), with convenient rest periods between. If less than 500 ml. are used for any one feeding, only the requisite amount should be put into the drip apparatus, and the remainder refrigerated until needed.

5. Administration during any one feeding should be continuous and at a reasonably steady rate. In order to control the rate of flow, we have used a gravity drip apparatus which is equipped with an adjustable needle valve at the air inlet.[3] Attached to the underside of this valve is a Mariotte tube, so that the mixture in the reservoir is gently and continuously agitated by a stream of air bubbles. The Wyeth intragastric drip apparatus has been found particularly useful for this purpose when the Mariotte tube is used. For complete mechanical control of the rate, such as was required in an investigation of long duration to be described later, we have used a special pump.† For a description of this pump see the Addendum.

6. Before the drip apparatus is connected to the jejunostomy tube, the latter should be flushed with sterile water from a syringe to make sure that it is patent. However, plugging of the tube with the aliment can be obviated if

*A radiographic study on jejunostomy patients of the rate of transport of the aliment away from the stoma has demonstrated that intestinal movement is sufficiently rapid one to two days following operation to permit of such early jejunal feedings with impunity.[4] This has been possible without deleterious consequences, even in the presence of an uppper abdominal peritonitis, provided all of the usual precautions are taken.

†Manufactured by the Marsh Precision Pump Company, Pittsburgh, Pa., and improved in collaboration with us. The authors are indebted to Mr. Jack C. Marsh for the use of this pump and for his cooperation in adapting it to the requirements of this study.

it is flushed immediately after each feeding. Of course, all air must be driven out of the connecting tubing of the drip apparatus before it is attached to the jejunostomy tube.

7. For orogastric feeding (for example, through a Levin tube) or for administration by gastrostomy, all these precautions should be taken—except that the rate may be increased considerably if desired.

8. The total water intake of an adult should ordinarily be 3 to 4 liters per day. Additional amounts of water over that afforded by the aliment may be given by means of the same drip apparatus between regular feedings; the rate may be greater than that for the aliment itself. Likewise, if the patient manifests any signs of a vitamin or mineral deficiency, therapeutic doses should be administered between regular feedings.

9. Successful use of this feeding mixture in the alimentation of patients with gastrointestinal fistula depends on the careful observance of the foregoing precautions. Disturbing symptoms usually result only from a failure in respect to these safeguards, and disappear when the proper technique is used. On the other hand, motor disturbances of the small intestine may be a consequence also of the patient's postoperative condition, since they are encountered after practically every type of major abdominal operation. When the latter is the case, the cramps, diarrhea, etc., persist even when the aliment is replaced by isotonic saline, or when intrajejunal drip is discontinued entirely. Under these conditions, tubal feeding may be continued at a slow rate in spite of the patient's distress.

### A CONTROLLED STUDY OF THE NUTRITIVE EFFICACY OF THE ALIMENT*

F. Z., male, aged 17 years at the time of this last operation, was under observation in this hospital for almost two years with cardiospasm unrelieved by surgery. Because of the continued inability of orally ingested food to reach the stomach, a jejunostomy for alimentation was performed. Following this, for a period of twenty months, the subject was maintained almost exclusively on the aliment described in this paper. During this time a study was being made of the influence of the jejunal intake of various nutritional factors on his voluntary food intake by mouth. Such a study was possible because the patient manifested a decided desire for ordinary foods by mouth, in spite of almost immediate and complete regurgitation from the esophagus. Repeated fluoroscopic study revealed that at most there was only a slow trickle of fluid into the stomach, and that solid food was excluded completely. Since the subject was ambulatory, he was transferred to the laboratory for controlled study during the day. He returned to his home at night, for week-ends, and even for a summer vacation of two weeks' duration. During such intervals, he was supplied with the requisite number of units of the aliment, which was stored at home and was self-administered according to instructions.

Usually, a patient in the immediate postoperative state never receives the aliment at a rate in excess of 200 ml. per hour. By the time this patient came under controlled observation, however (ten months after the jejunostomy operation and four months after a subsequent exploratory operation), the intestinal tract had developed a considerable tolerance to the jejunostomy feedings, and it was possible to start at a rate of 300 ml. per hour. At his own request this was gradually increased to a value about 700 ml. per hour, at which

*The two patients referred to in this section came from the surgical service of Dr. Harold Neuhof and the medical service of Dr. George Baehr, respectively. The authors acknowledge permission to study these cases.

level it was maintained routinely thereafter. Under such conditions, bowel movements were regular (once or twice a day) and the stools were generally well formed. On occasion, the rate was increased considerably above this level (as high as 2 liters per hour for a single feeding) for a short interval, but this sometimes resulted in abdominal discomfort, slight nausea, headache, and a diffuse feeling of warmth.

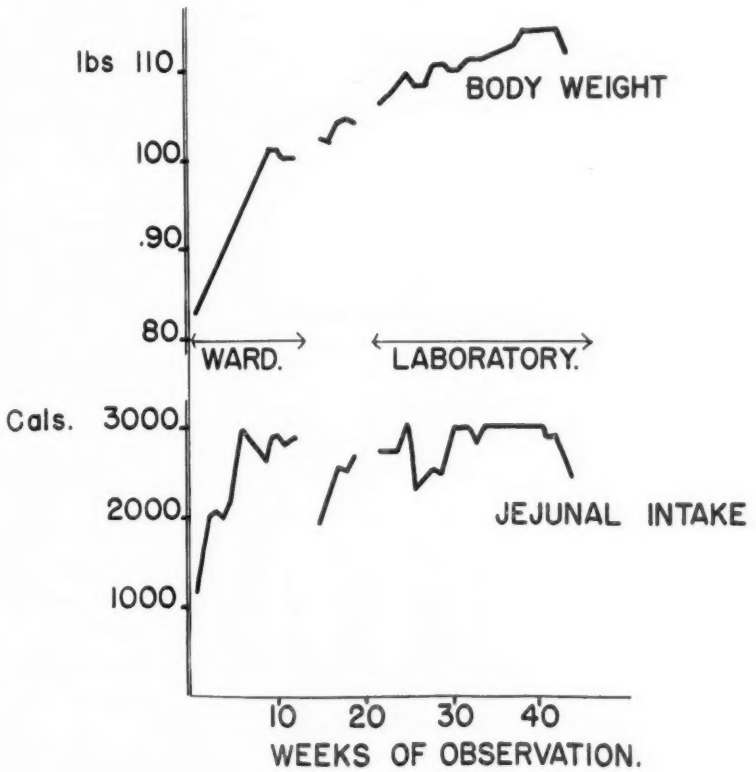


Fig. 1.—Weight and caloric intake curves showing maintenance on aliment MSH 47 alone. Patient F. Z., cardiospasm and jejunostomy.

At a caloric intake of about 3,000 Calories per day, during a period of increasing body weight, about 90 per cent of the nitrogen content of the aliment was absorbed. It is noteworthy, also, that the final concentration of the feeding mixture could be increased to 1.25 Calories per milliliter without causing any discomfort to this patient.

A detailed report of studies on this patient will be published later. For present purposes, however, we are concerned only with his improvement in body weight in response to jejunal feeding with this synthetic predigested aliment (see Fig. 1). At the beginning of the feeding with this preparation, shortly after the jejunostomy operation, his weight was approximately 70 pounds. From then until the beginning of the present study, it improved as long as he was maintained on the aliment in the hospital, but when he returned home and domestic feeding preparations were employed, the weight regressed characteristically. At the time of transfer to the laboratory for controlled study, his weight was 81 pounds. During the first forty weeks, no nutritionally significant changes in the character of the diet were introduced and his weight increased to a peak value of 116 pounds. Thereafter, experimental modifications in the diet were instituted, so that subsequent variations in body weight are not pertinent here.

S. L., female, aged 20 years, was a regular ward patient. The weight response of this patient with anorexia nervosa, who received the aliment for more than one month, is shown in Fig. 2. Weight prior to onset of symptoms was approximately 135 pounds. However, on admission her weight was 69 pounds and there was a marked nutritional edema. This girl's voluntary food intake failed to improve on oral feedings during the first three weeks of hospitalization, in spite of the usual therapies. At the end of this period, an indwelling nasal tube was passed into the stomach, and she was given varying amounts of the aliment by this route. Daily caloric intake of the aliment varied between 500 and 2,000 Calories. Only after this supplementation of oral intake with the aliment was initiated was a weight gain observed—from about 66 to 99 pounds. Concomitantly, voluntary oral intake increased and it became possible to initiate psychotherapy. The patient was discharged on the fifty-second day, at which time weight was 98 pounds.

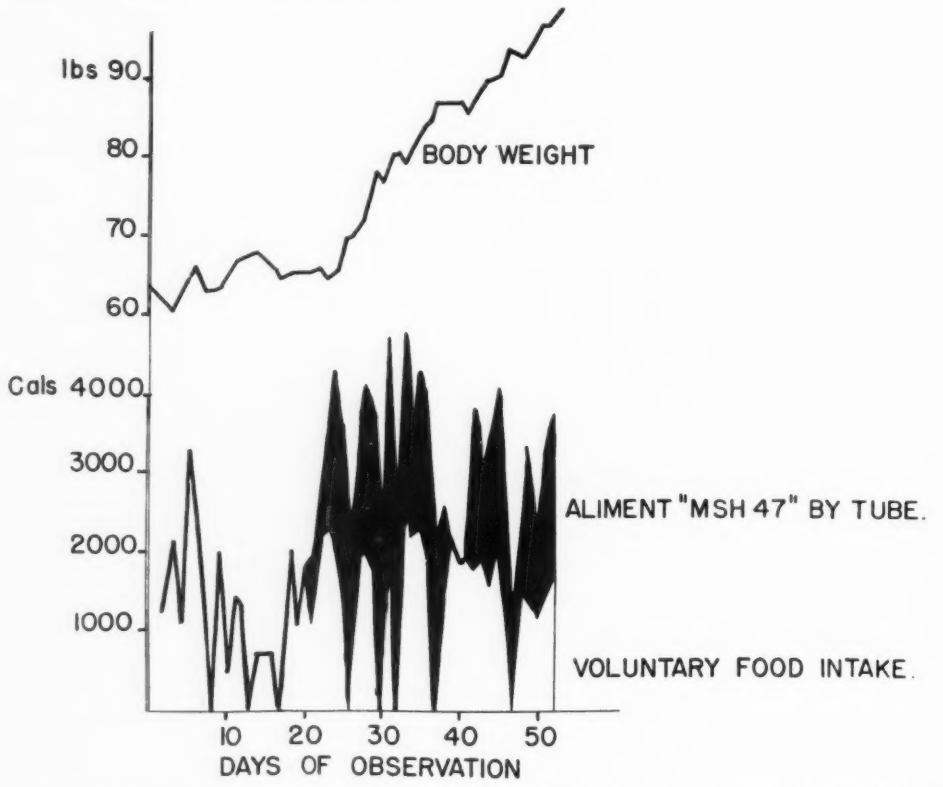


Fig. 2.—Weight and caloric intake curves showing voluntary food consumption supplemented by aliment MSH 47 via orogastric tube. Patient S. L., anorexia nervosa.

ADDENDUM

The action of the Marsh Precision Pump depends on the milking of the fluid through a rubber tubing (No. 4 in the schematic diagram, Fig. 3) by three fingers (No. 1, 2, 3) which compress the tube alternately. In this way, sterile material can be delivered to the patient with *no risk of its contamination* by the pump. Furthermore, the device is so constructed that it can control not only the *rate of flow*, but the *pressure of delivery* as well.

The diagram shows the relative positions of the three fingers at the beginning of a stroke, with No. 1 and 2 completely compressing the rubber tubing. These fingers are operated by cams driven through a gear train by an electric

motor. As the cams rotate, finger No. 2 moves upward and releases the tubing which then expands. This draws the fluid from the reservoir of the drip apparatus into the tubing as far as the outlet closed by No. 1. Finger No. 3 now advances, closes the inlet of the tubing, and remains in this position until No. 1 has been drawn upward to open the outlet. Following this movement, No. 2 (which is proportioned so that five drops are driven over) advances to force the liquid forward under No. 1. The latter then closes the outlet and No. 2 also descends, while No. 3 rises in preparation for the next stroke. Since at least one of the fingers is always in the down position, the tubing is closed at all times.

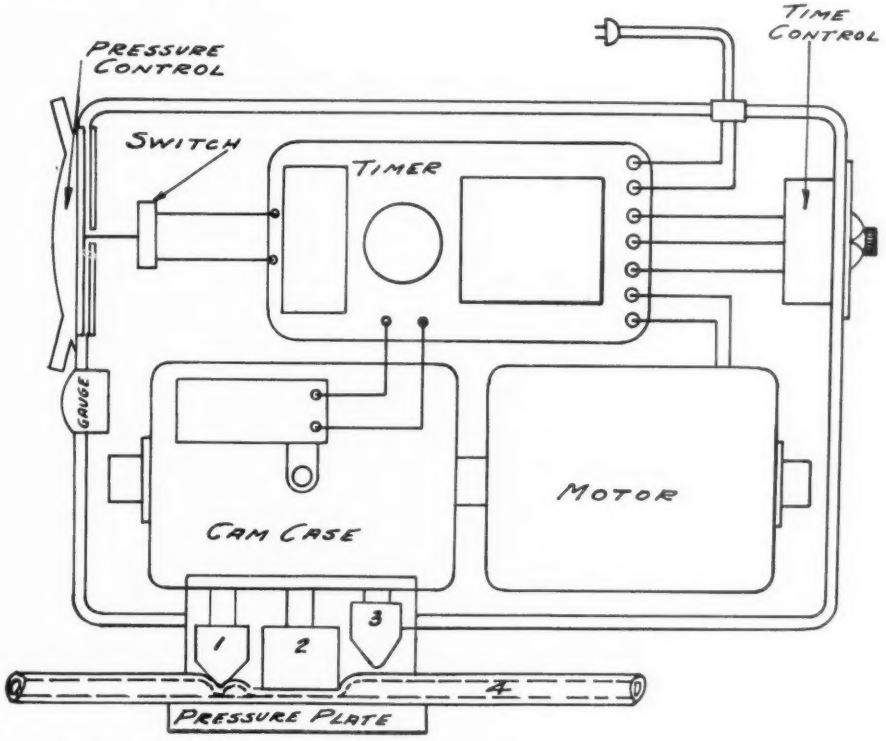


Fig. 3.—Marsh precision pump, schematic diagram (Model S—Pat. 2351828).

At no time does the fluid come in contact with moving parts, such as valves or pistons. In addition to maintaining sterility, this obviates both corrosion and clogging of the pump itself. The short length of rubber tubing may be sterilized before use and discarded after treatment if that seems desirable.

For the purpose of regulating the delivery rate, so that it can be adjusted accurately to the requirements of the patient, variable speed motors and gear transmissions were found to be unnecessarily complex. Instead, an electronic timer was developed, which allows the pump to deliver a single stroke (5 drops) and then stop for a time. This "wait" can be adjusted from less than 1 second to more than 60 seconds, before the next stroke is made.

Thus, the average delivery rate of the pump is easily fixed by setting a pointer on a calibrated scale.

Delivery pressure is controlled by a three-inch diaphragm in a sealed system, through which the fluid passes (not shown in the diagram). The diaphragm and face cover are constructed so that they can be dismantled and easily removed from the system for sterilization by autoclaving. The pressure gauge and the manual adjuster for setting the pressure, however, do not come in contact with the sterile fluid at any time. Such adjustments are effected by a mechanical connection at the back of the diaphragm. An automatic switch is incorporated into this control, so that the motor is automatically shut off whenever the desired pressure is reached, and is started again only after the pressure has been relieved. The gauge is of a standard type, calibrated in inches of water; it is placed so that its face indicates clearly the pressure at which the aliment or other drip fluid is being delivered.

The entire pump is housed compactly under a plastic cover 6 by 7½ by 9 inches, which is provided with a handle. Its relatively light weight makes it easily transportable.

## SUMMARY

This report describes a modification of the synthetic nutritive mixture, devised in this laboratory several years ago for the feeding of patients with jejunostomy. The original aliment was designed to supply all the nutritional elements required to maintain surgical patients for two or three weeks, either prior or subsequent to other major surgery of the digestive tract. Salivary and frequently gastric digestion are completely eliminated in such cases, as a result of a high obstruction, and pancreatic digestion is considerably reduced by reason of the level of the small bowel at which the feedings are administered. Hence, this synthetic foodstuff is prepared in a predigested state, by the use of partially hydrolyzed protein and carbohydrate. The fat content is kept very low. The major part of the carbohydrate is in the form of dextrins rather than disaccharides; monosaccharides are virtually absent. As a result of these restrictions, and also because of a suitable dilution with water, administration of this aliment does not induce the motor disturbances (for example, cramps, vomiting, and diarrhea) which are commonly associated with jejunal feedings of natural foodstuffs—provided the rate of administration is suitably controlled, and adequate attention is given to maintenance of sterility. When such disturbances do occur, in spite of these precautions, they usually persist even when isotonic salt solution is the sole article of jejunal diet. This demonstrates that the excessive motor activity is not caused by the feeding mixture but is a consequence of the surgery itself, and often follows major abdominal operations. The NaCl content of the aliment is somewhat higher than is ordinarily necessary, but this was designed in order to restore and maintain water and electrolyte balance in patients with gastrointestinal obstruction.

The present modification of this jejunostomy aliment follows the general pattern of the earlier form, but has the following additional advantages:

1. Its nutritional quality, particularly in regard to the more recently discovered vitamins, has been greatly improved so that the preparation can be used as a sole source of nutrition for long periods of time (for example, in patients with permanent or semipermanent jejunostomy or gastrostomy).

2. The mode of preparation has been changed so that large amounts can be prepared at one time and packaged in 500 Calorie units, thus making it possible to keep an adequate supply on hand at all times.

3. The material can be kept for at least four weeks without bacterial growth or significant loss of vitamin potency, provided the containers are not opened until required for use.

4. The aliment can now be supplied for self-administration at home.

This jejunostomy aliment has found many uses in other than patients with jejunostomies, for example, those with gastrostomies, those requiring orogastric tube feeding, etc. At the discretion of the physician, the total caloric concentration as well as the content of vitamins and protein hydrolysate can easily be varied, although the latter is considered to be about optimal for most purposes. Stocks of the salt mixture, vitamin mixture, and fat emulsion are prepared in the pharmacy, but the complete aliment is made up in the diet kitchen. A special pump is described for use in situations requiring complete mechanical control of the pressure and rate of administration.

The efficacy of this diet for long-range feeding is demonstrated by a report on a patient with cardiospasm unrelieved by surgery, who has a jejunostomy for feeding purposes. This individual was maintained on the aliment as a sole source of nutrition for more than one and one-half years, during which time his body weight increased from a low of 71 pounds to a maximum of 116 pounds. Nitrogen balance studies revealed about 90 per cent efficiency in absorption of the protein hydrolysate, which may be ascribed to the predigested character of the preparation. The successful use of this aliment for orogastric feeding in a patient with anorexia nervosa is also reported.

The authors wish to acknowledge their obligations to the following companies for their generous supply of materials used in this work: Merck and Company, Inc., for Biotin; Distillation Products, Inc., for the mixed tocopherol concentrate; Wyeth, Incorporated, for the protein hydrolysate, Lactamin; Wilson Laboratories for whole liver substance; and Lederle Laboratories Division, American Cyanimid Company for inositol and pteroylglutamic acid (Folvite).

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# EXCISION OF SMALL TUMORS OF THE SKIN OF THE FACE WITH SPECIAL REFERENCE TO THE WRINKLE LINES

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AFTER thought has been given to the adequacy of removal of a lesion on the face, the next most important consideration is the selection of appropriate lines for excision so that the ultimate scar will not interfere with function and will be as inconspicuous as possible. For almost ninety years surgeons have been referring to Langer's lines as the most appropriate for the direction of incisions on the body. However, experience in surgery of the face leads inevitably to the conclusion that these are not the most suitable. It is the purpose of this paper to describe the formation of normal wrinkles, to point out how they guide one in planning surgery on the face, and to outline some useful refinements in surgical technique.

## LANGER'S LINES

Historically, Langer's lines are of considerable interest. The original article describing them appears in the *proceedings of the Society for Natural History of Vienna* in 1861. In this paper, Langer[1] referred to an observation made by Dupuytren that, in a case of suicide, the round holes in the skin made by an ice pick were drawn into ovals. The fact that there are lines of tension in the skin therefore was recognized in the earliest days of modern surgery. Langer's observations were based on post-mortem studies. The cadavers were those of patients who had died during early adult life and also during the advanced age decades. Cutaneous punctures were made by the introduction of an awl (2 mm. in diameter) to a point of 2.5 cm. below the surface of the skin. His determinations were made on skin in a static condition and his hypothesis was that the elliptical distortion of the puncture wounds was produced by the tension of minute rhomboids of connective tissue fibers in the skin. These rhomboids are presumed to be always present in unbroken skin and are arranged somewhat like geodetic lines on the earth's surface. Doubt was cast on the suitability of incisions following Langer's lines for the most satisfactory healing of abdominal wounds by one of us,[2] who observed that scars of oblique incisions over the lower abdomen became wider in their lower portions. It was pointed out that the lines of elasticity of the skin run transversely in the suprapubic region rather than obliquely vertical as described by Langer. The suggestion was made that the physiologic elasticity of the various surface regions may be different in the living subject.

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Photographs and sketches prepared by Department of Medical Illustration, U. S. Veterans Hospital, Bronx, N. Y.

Received for publication, July 20, 1948.

This discussion will be limited to the face, although Langer's illustrations show the lines of tension over the entire surface of the body. Fig. 1 is a reproduction of Langer's original plate of the side of the face showing how the punctures develop into ovals. Lines have been drawn lightly through their long axes. The triangular areas represent points at which the projected lines meet from three directions. These lines are reproduced in many publications on surgery[4-8] with present-day surgeons commenting that "incisions closed parallel to these lines will be less likely to cause wide scars than those across

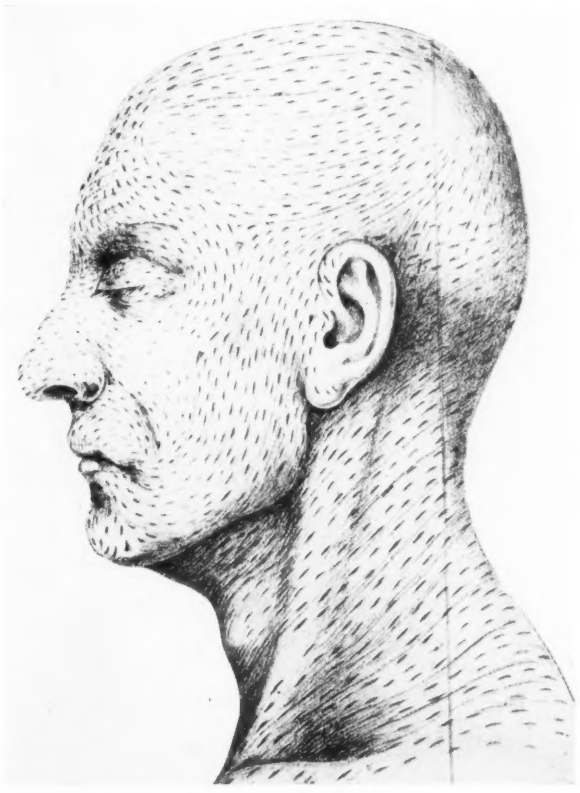

Fig. 1.—Reproduction of Langer's original diagram of the face indicating how he arrived at the lines of skin tension. (From Langer,[1] 1861.)

them"[4] and "incisions made along these cleavage lines will afford good exposure and result in minimum scarring as wound edges fall naturally into advantageous positions, thus reducing tension on wound margins and stress on sutures."[6] If one considers Fig. 2 which has been reproduced from Kirschner and Shubert's *Surgery*,[3] it is at once evident that there are areas where one would rarely make an elective incision following Langer's lines. The most conspicuous of these areas are in the supraorbital region, on the side of the nose, in the lateral ocular region, lateral to the angle of the mouth, on the upper lip, on the lower, outer aspect of the chin, and in the malar region.

NORMAL WRINKLE LINES

What then are the ideal lines for planned surgery on the face? If one observes Figs. 3 and 4 the answer becomes apparent. These illustrations were produced by photographing an old man. This subject was selected because the facial skin was especially loose. Photographs were taken in repose and after contracting the muscles of facial expression. These photographs were then compared with other subjects and a composite sketch was made by the medical artist. The illustrations exaggerate the normal wrinkles and serve to indicate the direction of the lines of relaxation of the skin. If a facial incision is planned so that it is parallel to these lines, the scar will be relatively inconspicuous. These lines are the guides which plastic surgeons use, often referring to them erroneously as Langer's lines.

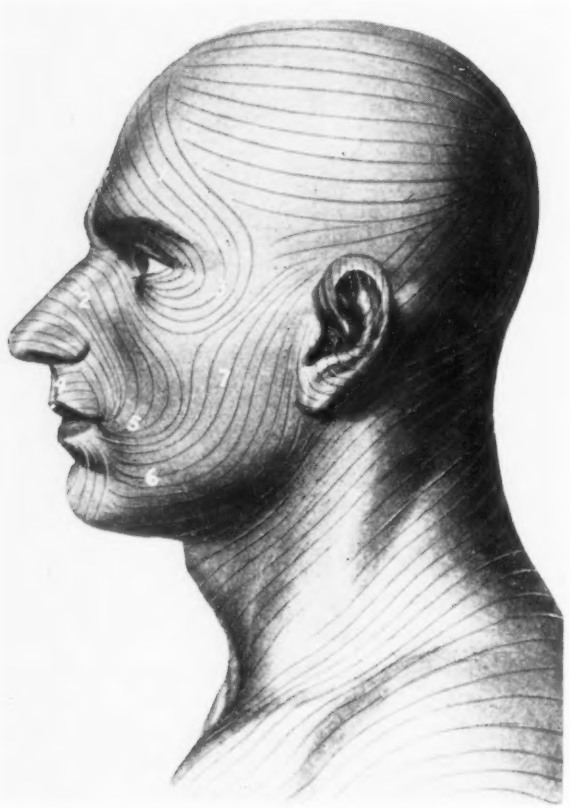


Fig. 2.—A reproduction of Langer's lines on the face. Areas where one would rarely make elective incisions following these lines are indicated by the numerals. These are discussed in the text. (From Kirschner and Shubert,[3] 1927.)

If one considers the mechanism of the formation of facial wrinkles, it will be appreciated that they are produced by the contracture of the underlying muscles of facial expression. The simplest example is the transverse corrugation in the forehead caused by the contraction of the frontalis. In some regions, however, several muscles act in unison to produce facial expression with

the result that the skin wrinkles in a curved line. A conspicuous example is the nasolabial fold which is the result of the action of the zygomaticus, the quadratus labii superioris, and the caninus. The risorius and buccinator also may play a part in the production of this very definite nasolabial groove. Likewise one may go over the entire face and find the underlying muscles of facial expression which through their contraction develop wrinkles at right angles to direction of their pull. It is evident that the wrinkles are caused by the shortening of the muscle without corresponding shortening of the skin. Therefore, the skin adapts itself by forming folds which are at right angles to the actions of the underlying muscles in that particular region. In the

Fig. 3.

Fig. 4.

Fig. 3.—Side view of face; a composite drawing of normal wrinkles.
Fig. 4.—Front view of face; a composite drawing of normal wrinkles.

infraorbital region this hypothesis does not seem to be true, as the wrinkles are in the direction of the fibers of the orbicularis oculi. This may be explained by the levator action of the orbicularis in the lower eyelid. Attention is called to the fact that the sphincteric action of the orbicularis oculi is altered by its origin and insertion at two fixed points, the palpebral ligament at the inner canthus and the lateral palpebral raphe at the outer canthus. Contraction of this portion of this muscle results in lines at right angles to the direction of its fibers and the wrinkles then are at right angles to the action of the muscle. At the upper portion of the dorsum nasi, the corrugator

supercilii and the orbicularis oculi act somewhat antagonistically to the frontalis to form the almost vertical lines observed during a frown. In the oral region, the orbicularis oris, being a true sphincter, has no fixed points and therefore the wrinkles develop radially from the oral aperture. However, at the angles of the mouth, the combined action of the quadratus labii superioris and the other muscles in this region causes the lines to blend with those of the lower part of the nasolabial fold. The formation of the lines about the outer aspect of the chin is complicated as the wrinkles in this area are the result of the action of the triangularis, the depressor labii inferioris, and the mentalis. To illustrate the position of these lines Fig. 5 was drawn showing their relation to the underlying muscles.

Fig. 5.—The black lines in this figure are tracings of the wrinkles shown in Fig. 4 superimposed upon the muscles of facial expression. Note that these wrinkle lines uniformly are at right angles to the direction of contraction of the muscles.

Many younger subjects do not have evident wrinkles but careful inspection of the skin usually will reveal fine lines of cutaneous relaxation. In order to exaggerate them, one need only ask the patient to contract the muscles in the region concerned and the lines become apparent. All subjects are not precisely the same and no single chart of facial wrinkle lines will serve for all cases. One cannot generalize quite as freely as would be indicated by Langer's lines, but consideration of the anatomy of the muscles

of facial expression makes it possible to develop a fairly definite pattern for the planning of incisions on the face.

## ADEQUACY OF EXCISION

In the surgical excision of tumors it is essential to remove the lesion in its entirety and the first consideration should be given to the adequacy of this excision. Plastic surgeons have been critized for placing undue emphasis on the closure which will give the most satisfactory cosmetic result. On the other hand, needless mutilation may occur in the surgeon's overanxiety to remove all of the diseased tissue or in his failure to respect the normal wrinkle lines. The surgery of facial tumors requires knowledge of and experience in cancer surgery and also knowledge of the principles of plastic surgery. At the time of primary excision the surgeon has the best chance of eradicating a malignancy completely. It is the responsibility of the surgeon who first treats the patient to allow adequate margins in the excision of the tumor. The opinion of an experienced surgical pathologist, whose advice is based on microscopic study of a biopsy of the tumor, is sought wisely before the primary excision rather than after the development of a recurrent tumor.

To give a general conception of what might be considered adequate, the counsel of such an experienced surgical pathologist was sought. He[10] suggested that the following margins could be used as a guide though each case should be considered individually. These values apply only to the management of small tumors of the skin which have not invaded subcutaneous tissue. Their listing is intended only as a guide. Nevi, pigmented or nonpigmented, require a margin of only 2 to 3 mm. Basal-cell carcinomas of the skin may be classified by inspection as of the elevated or burrowing type. The margin of excision of normal skin around the former should not be less than 0.5 cm. For the burrowing lesion it should not be less than 1.0 cm. However, squamous-cell carcinomas require at least 1.5 to 2.0 cm. for safe lateral margin. The depth of the excision also is extremely important. Logically, one should excise as deeply as one does widely. In practice the deep fascia if not invaded by tumor is a good limiting membrane and if removed with the lesion this should be considered sufficient depth of excision for malignant tumors of the skin. Malignant melanoma should be given a wide margin of excision, certainly not less than 3.0 cm. beyond the margin of visible pigmentation and palpable tumor. We are not in agreement with those who advocate very wide (8.0 to 10.0 cm. margin) excision of regional skin in attempt at cure of the tumor.

Whether or not the removal has been adequate can be determined finally by the pathologist's examination of the specimen. Longitudinal and transverse sections should be made with report on the width and depth of normal tissue still attached to the lesion.

## TECHNIQUE OF EXCISION

After the patient has been prepared and anesthetized one may use a marking fluid, such as gentian violet, to indicate the lines of excision. Dots are made around the lesion, all equidistant from the margin of the tumor. An ellipse,

perfect or irregular in outline, is then marked out so that it includes the dotted area and yet has its long axis in the direction of the wrinkle lines (Fig. 6). The length of the ellipse should be at least four times as great as its width. The longer the length as compared with the width the less likelihood of "boating" of the tips of the line of suture. The lines should be drawn on the skin so that both sides of the ellipse will be approximately the same length. If this is done, adjustments will not have to be made at the margins of the wound. If not possible, adjustments of the marginal tissue must be made. Referring to the area beneath the left eye in Fig. 6, if the ellipse was made from *a* to *d* directly, the lower line of excision would be too long to approximate accurately to the upper line. One therefore selects a point *c* which should make *ac* equal to

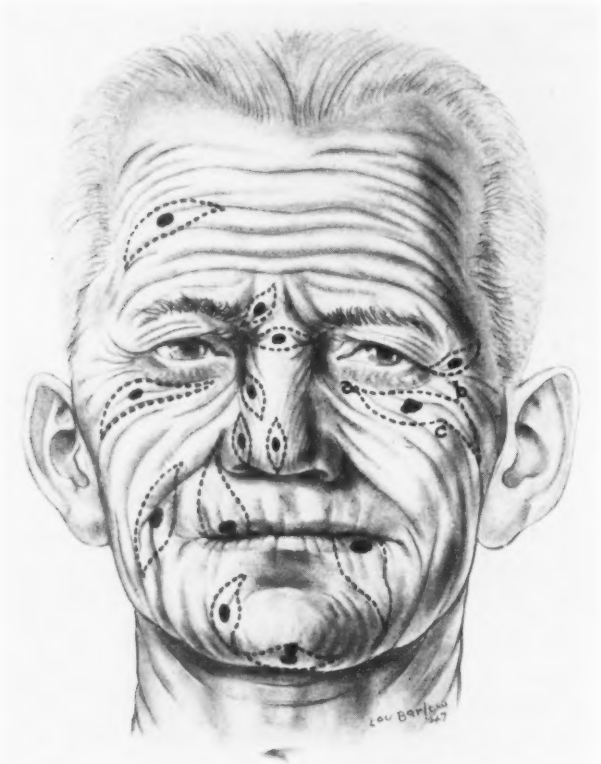


Fig. 6.—Diagram to show suggested lines of excision to allow ultimate scar to fall in normal wrinkles.

*ab*. The small triangle *b-c-d* may be removed with the result that the incisional margins will have equal length and the line of suture will fall in the wrinkle lines. This principle may be applied to other regions of the face where the same problem is present. Usually the wrinkle lines present as gentle curves. In making skin incisions, effort should be made to keep the scalpel perpendicular to the skin surface in order to prevent the beveling of cutaneous margins. The mass should be removed en bloc to avoid disseminating tumor cells. After the lesion has been removed, wide undermining of the skin and subcutaneous tissue

must be done to allow relaxation. A useful plan is to undercut the margins of an elliptic defect for a distance as great on either side as the transverse measurement of the ellipse. The use of 00000 and 000000 chromic catgut has proved to be satisfactory for approximating the subcutaneous tissue and the derma. Sutures in the derma must be placed accurately so that the suture material does not lie in the epithelium and the knots should be on the underside. Sutures placed close to each other will effect closure of the skin, making it necessary to use only the finest sizes of silk for suture of the surface of the wound. If buried in the derma, silk suture may protrude through the scar at a later date, even though no infection has occurred. Cutaneous sutures should be removed on the third or fourth day after operation. In the postoperative dressing of large wounds a layer of xeroform gauze overlayed with several thicknesses of plain gauze is held in place with adhesive, applied tightly enough to take

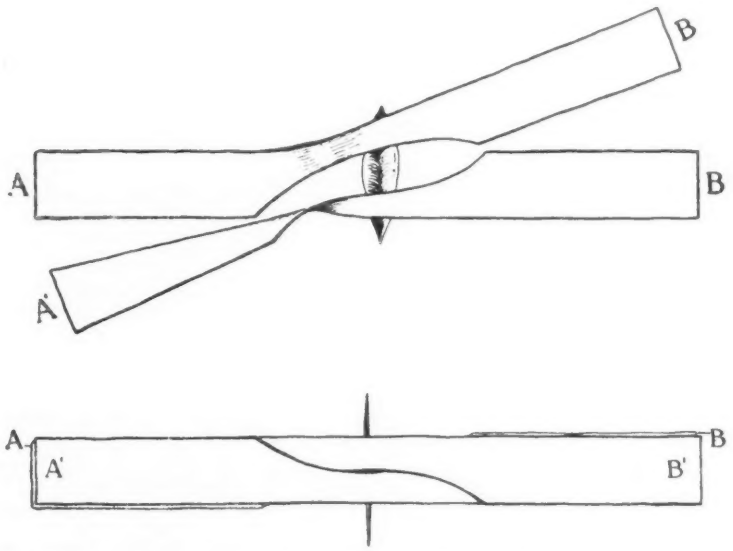


Fig. 7.—Diagram of approximation of wound margins by flamed adhesive. Apply ends *A* and *B* on opposite sides of the wound after flaming the central portion of the strips. Draw wound margins together by pulling *A* and *B* in opposite directions. Apply *A'* over *A*, and *B'* over *B*, locking strips when wound margins are together. (From Kraissl,[9] 1933.)

tension off the suture line. Hematoma may be prevented and ecchymosis diminished by the use of cotton and elastic bandage applied with even pressure. After the sutures have been removed, flamed adhesive may be applied, similar to the method[9] of approximating wound margins as indicated in Fig. 7. Smaller wounds are effectively managed by the crepe lisse dressing[2] which consists of silver or aluminum foil over the sutured wound, held in place by a single layer of indestructible chiffon which has been immersed in collodion. The setting of the collodion provides an effective splint for the skin of the face so that involuntary facial expression does not cause movement of the sutured tissue. Sutures may be removed through a V-shaped gate cut in this cutaneous splint of collodion and chiffon. The splint may be left in place for seven or eight days.

## CONCLUSIONS

1. Langer's lines, although copied and referred to by surgical texts, are not the most suitable for planning the direction of incisions on the face.

2. The normal wrinkle lines which are at right angles to the action of the underlying muscles of facial expression afford the best guides for incisions on the face.

3. Complete removal of tumors of the skin of the face by careful surgical technique with attention to the normal wrinkle lines is rewarded by superior clinical results with minimal deformity from facial scars.

The authors wish to express their sincere appreciation to Mr. Lou Barlow, Medical Artist of the Veterans Administration Hospital for his excellent illustrations.

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*Addendum:* Since the preparation of this manuscript, it is noted that Rubin came to the same conclusions regarding Langer's lines and facial scars using a police detection device for skin line study. (Rubin, Leonard R.: Langer's Lines and Facial Scars, Plast. & Reconstruct. Surg. **3**: 147-155, 1948.)

# A SIGMOIDOSCOPE WITH PROXIMAL AND DISTAL ILLUMINATION

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THERE are usually two broad types of sigmoidal endoscopes, namely, those that are provided with distal sources of illumination and those with proximal illumination. Those endoscopes with distally placed light provide excellent illumination which, however, frequently becomes dimmed by adherent particles of feces or excretions. These difficulties are avoided by the use of an endoscope that has the light placed at its proximal end. However, owing to diffusion of the light, proximal illumination is not as bright as the distally lighted instruments. Realizing the advantages of each type of illumination, I have designed a sigmoidoscope that is provided with proximal and distal lighting systems that can be employed simultaneously or singly, and hence incorporates the advantages of both systems of illumination.

The instrument consists of tubular shafts or sheaths made of light metal, bakelite, or Lucite, varying in length from 2 to 10 inches (5 to 25 cm.) and ¾ to ⅞ inch (about 2 cm.) in width (Fig. 1). The distal end of the shaft is slightly slanted, rounded, and smooth to facilitate the easy introduction of the instrument into the anal outlet and to prevent mucosal abrasions. The proximal end of the shaft has a wide circular rim to which a handle is attached with ease (Fig. 2, *A*). A connection for the electric current to all sources of illumination as well as a switch for the control of the distal sources of illumination are situated at the free end of the handle (Figs. 1 and 2, *B*). The opposite end of the handle (near the opening of the tubular shaft) is capped by a proximal source of illumination (Figs. 1 and 2, *B*) which consists of a metal case containing a strong light bulb. This light bulb is activated when it faces the opening of the tubular shaft and is turned off by merely swiveling the light carrier to either side. Diffusion of this proximal light is prevented or greatly reduced by a movable circular metal sheath which can be extended to the opening of the tubular shaft (Figs. 2, *B,* note arrows, and 3).

A metal cap containing a magnifying lens fits snugly into the proximal opening of the shaft (special telescopes are unnecessary). This cap also is used to close the tubular shaft hermetically when air insufflation is desired for distention of the bowel. Through an inlet situated on the right side and close to the proximal end of the shaft, air is introduced by means of a bulb which has a Luer locking device (Fig. 3). The bowel may be deflated either by removing the inflating bulb or more expeditiously by opening the suction outlet (Figs. 2 and 3).

The suction tube is situated in the interior of the shaft resting on its floor, and is terminated at its distal end (Fig. 2). It may be employed for the in-

Received for publication, July 19, 1948.

troduction or removal of fluid and for the withdrawal of smoke resulting from fulguration. Through this suction tube, culture media may be injected and withdrawn with the aid of a sterile syringe (Fig. 2), thus obtaining washings of intestinal flora for bacteriologic studies.

The instrument as developed to this point is a practical, simple, and useful unit for diagnostic and therapeutic purposes even in the hands of the occasional operator or of the nonspecialist in coloproctologic diseases.

Fig. 1.—The dissembled sigmoidoscope and anoscope. *1*, Shaft of sigmoidoscope showing the suction outlet and the inlet for insufflating air, *2*, Magnifying lenses; *3*, Holder or carrier of distal lights; *4*, handle with proximal light facing anoscope, circular switch, and connection for electric current at opposite end.

Additional refinements have been developed that can be added conveniently to the sigmoidoscope just described. Brighter illumination may be obtained by the introduction of a light holder or carrier into the interior of the tubular shaft along side the suction tube having one light bulb placed at the distal end of the shaft and another light placed about 4 inches (10 cm.) above this point (Fig. 1, *3* and *2*). This light holder fits snugly into the proximal opening of the shaft and establishes a specially provided contact with the handle for transmission of the electric current. The distal lights are con-

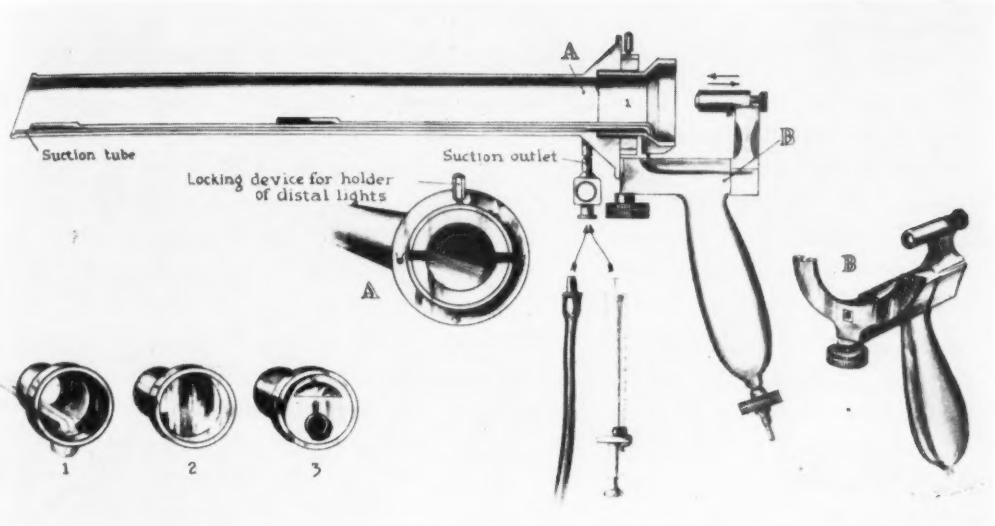


Fig. 2.—Showing the interior of sigmoidoscope and the distal light carrier in situ. *A* shows wide rim to which the handle (*B*) is attached as well as the locking device for distal light holder. Arrows over proximal light carrier indicate the travel of the sheath used for the control of diffusion of the proximal light.

Fig. 3.—Sigmoidoscope assembled with both systems of illumination, the magnifying lens, and rubber bulb used for insufflation.

trolled by the switch. One of the distal lights can be lit simultaneously with the proximal light so that two sources of illumination may be utilized simultaneously.

The magnifying lens previously described can also be used with the light holder in situ. A still more refined and useful feature is a metal cap, one-half of the circumference of which is occupied by a magnifying lens, while the remaining one-half contains an opening surrounded by a washer which makes possible the snug passage of a biopsy forceps or an electrode (Figs. 1 and 2, 3). This facilitates the visualization during the removal of small mucosal excrescences for microscopic study and their subsequent destruction by desiccation.

## SUMMARY

A colorectal endoscope is described which has one proximal and two distal sources of illumination. The proximal and one of the distal sources of illumination may be used simultaneously. Either lighting system can be operated independently.

A suction tube is incorporated in the tubular shaft through which fluid can be introduced or removed, smoke incident to fulguration may be withdrawn, and culture media may be introduced and withdrawn, thus obtaining intestinal washings adequate for cultural studies.

# ANTIHISTAMINE THERAPY IN THROMBOPHLEBITIS MIGRANS

## A Case Report

Frank A. Selecman, M.D., and Edward W. Miller, M.D., Dallas, Texas

THE purpose of this paper is to report one case of thrombophlebitis migrans in which the course of the disease was apparently halted abruptly by the use of one of the new antihistamine drugs.

No attempt is made to review the literature in any detail, but the commonly accepted theories of etiology will be mentioned, and a useful bibliography appended. An excellent recent review of the literature with a discussion of the disease in some detail and the presentation of six cases may be found in the article by Fischer published in 1946.

Thrombophlebitis migrans is a disease of the young and middle aged which involves the deep and superficial veins, particularly the small and medium sized veins, but not infrequently the larger venous trunks in an acute nonspecific inflammatory process which results in occlusion of the vessel so involved. The disease is characterized by chronicity and a tendency to recur, and its seriousness depends on the mechanical impediment of venous flow which results from the occlusions of the blood channels, rather than on the inflammatory reaction which accompanies the thrombotic process. The disease is most serious when it extends to involve the veins of the brain, liver, heart, lung, or intestine. Its migratory character and tendency to involve short segments of veins in scattered regions of the body, its failure to respond to the methods which may halt the localized venous thrombosis seen particularly in the veins of the legs, as well as its tendency to occur in the absence of the usual conditions which set the stage for venous thrombosis mark it as a clinical entity.

Sir James Paget in 1866 characterized it aptly as a type of thrombophlebitis which is not associated with the usual conditions favoring venous thrombosis such as trauma, exhaustion, infection, varicosities, local inflammation, and puerperium, and thought it associated with a tendency toward gout. Theories of toxic origin or of focal infection have not withstood the test of time.

Treatment has been quite unsatisfactory. Vaccines, dicumarol, heat, sulfonamides, penicillin, and heparin have been used with poor results; vein ligation, even if possible, does not stop the course of the disease.

Mortality figures range from 8 to 50 per cent.

It is for these reasons, then, that we thought it expedient to report an experiment with a new type of therapy in the hope that its effectiveness might be investigated by others.

### CASE HISTORY

A 50-year-old white, married woman was admitted to the Dallas Methodist Hospital on Dec. 23, 1947. The chief complaint on admission was aching pain in the left leg. The

Received for publication, July 26, 1948.

illness had begun ten to twelve days before admission, when the patient had noticed some pain in the lower left leg while standing and some aching in the left thigh. She had also been conscious of a low-grade temperature which she had checked herself at home. She had complained of an upper respiratory infection with a sore throat for the preceding two weeks.

*Past History.*—Twenty-five years before admission, following childbirth, she had developed a phlebitis of the lower right extremity which was of severe degree, but left her with no permanent edema. Following this initial attack there were numerous recurrences of low-grade phlebitis on that side. Ten years later, following perineal repair and appendectomy, she developed a marked flare-up of the vein infection on the same side. In the next few years there were several minor recurrences of the disease which she treated at home with bed rest and elevation of the leg. Three and one-half years before admission she was in apparently good health when she suddenly developed a recurrent phlebitis which was treated with sulfonamide therapy. The disease progressed, however, and she had at least one rather severe pulmonary embolism. Following this episode she underwent right saphenous vein ligation, at another hospital. She was kept in bed for a period of three months, during which time the phlebitis spread to involve the lower left extremity as well. Heparin therapy at this time failed to halt the progress of the disease, which gradually burned itself out, and she was sent home to remain in bed there for another three months. When again ambulatory she noticed that marked collateral vessels had developed in a network running from the thighs and the region of the groin upward over the lateral and anterior abdomen to join the superior vena cava return. After that severe attack she wore elastic stockings, ace bandages, and noticed numerous recurrences of pain in the leg, tenderness over the superficial veins, and muscle cramping. She noticed that these outbreaks of the disease were likely to follow upper respiratory infections, many of which she treated at home with sulfonamide taken by mouth. Past history is otherwise unremarkable and family history of no interest.

*Physical Examination.*—Examination revealed a moderately obese woman who appeared in good general condition. There was slight injection of the pharynx, the chest was clear, heart sounds were normal, blood pressure was 108/74. There was bilateral calf tenderness, tenderness over the left femoral vein which could be palpated as a firm cord, and there were marked collateral venous channels over the anterior abdominal wall and the region of both thighs. Measurements of the circumference of the lower extremities revealed no asymmetry. Temperature on admission was 99° F., white blood count was 10,000, hemoglobin was 13.6 Gm., the urine was not remarkable.

*Treatment.*—She was treated with bed rest, elevation of the legs, cradle light, and heparin intravenously at eight hour intervals. She had a low-grade temperature for the first few days; she then began gradually to return to normal. The heparin was discontinued on the third day because of phlebitis at the point of injection. After two weeks in bed she was gradually allowed to become ambulatory, at which time a large collateral vein approximately 1½ to 2 cm. in diameter was seen to extend from near the symphysis upward over the anterior abdominal wall to join other smaller veins over the sternum. She was discharged improved on the nineteenth hospital day.

*Second Admission.*—The patient was readmitted to this hospital on Jan. 26, 1948, with a recurrence of the disease. She had been treated at home during the interval between admissions with intramuscular heparin 200 mg. daily. Temperature on this admission was 99.2° F., urine was negative, white blood count was 9,400, with 15.4 Gm. of hemoglobin. Clotting time the day after admittance was 1 minute and 42 seconds.

Physical examination was much the same as on the previous admission, with the additional finding of tenderness and erythema over many of the collateral venus channels on both sides as well as over the large abdominal vein, previously mentioned. Many of the vessels could be palpated as firm, tender, fibrous cords beneath the skin. The patient was quite uncomfortable during this admission and careful observation showed quite clearly the daily spread of the disease. The cephalic vein in both arms became involved

as well as many of the smaller tributaries to the brachial veins. She was treated with bed rest and heparin by continuous drip intravenously for a period of many hours with no apparent benefit. Following this, heparin was given every eight hours in doses of 100 mg. intramuscularly for a period of eight days, with no obvious signs of improvement, therefore heparin therapy was discontinued. Clotting times over this period averaged from 5 to 7 minutes. Other laboratory findings included a negative blood culture, normal glucose tolerance curve, a bromsulfalein retention of 24 per cent in 30 minutes, a one plus cephalin flocculation, prothrombin level of 59 per cent. A cold agglutinin test gave negative results. X-ray studies of the chest and upper gastrointestinal tract and barium enema revealed no obvious pancreatic lesion. On the fifteenth hospital day the patient was started on Histadyl in doses of 50 mg. t.i.d. and 100 mg. at bedtime daily. Within twenty-four hours after the beginning of this therapy she noticed marked improvement in the general condition with reduction of the tenderness over the involved veins and disappearance of aching in the legs and lower abdomen. The patient was rapidly made ambulatory and seven days following the institution of Histadyl therapy she was dismissed from the hospital with a normal temperature and in excellent spirits, entirely relieved of any signs or symptoms of venous inflammation. She has been entirely well in the four months since she was discharged.

As consultants, the patient was seen by Dr. Carl Moyer, Dr. Robert Sparkman, and Dr. Howard E. Heyer, who felt that allergy might be playing some part in the disease.

To our knowledge this is the first case of thrombophlebitis migrans which has been treated, and treated apparently successfully, by an antihistamine drug.

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# MULTIPLE AORTO-INTESTINAL FISTULAS SECONDARY TO SWALLOWED FOREIGN BODY

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A CASE of aortoduodenal fistula secondary to a swallowed needle was recently encountered. The fistula was successfully closed without removal of the foreign body. Later it again caused a perforation from the aorta into the intestine through a false aneurysm, on this occasion at a lower level. The second fistula was also successfully closed and a part of the foreign body removed.

Arterio-enteral fistula is rare. In 1943 Bank[1] reported a case of aortoesophageal fistula which resulted from a swallowed fishbone. In his review of the literature he found four similar cases. In 1946 Dick and Nevill[2] added another case. All these cases were fatal and diagnosis was made in each case at autopsy. Bank[1] also found that sixty-three cases have been reported of rupture of great vessels other than the aorta as the result of a foreign body in the esophagus.

Aneurysms of the abdominal aorta are not uncommon. Occasionally they rupture into the gastrointestinal tract, producing massive hemorrhage. In a review of the literature by Rottino in 1943,[3] 32 cases were found in which an aneurysm of the aorta ruptured into the gastrointestinal tract. The stomach was perforated in 5 cases, the jejunum in 1 case, the duodenum in 24 cases, and the small intestine (site not specified) in 1 case. All the cases in this group were fatal, diagnosis having been made at autopsy.

In an extensive search of the literature no reports could be found of established fistulas between the abdominal aorta and gastrointestinal tract with or without an associated foreign body.

## CASE REPORT

R. W. D., a 21-year-old white man, was admitted to the medical service of the University of Virginia Hospital on Aug. 13, 1947, because of gastrointestinal tract bleeding.

At the time of admission to the hospital the patient gave a six weeks' history of intermittent episodes of sudden onset of weakness, dizziness, and nausea. These episodes were followed in several hours by the passage of large amounts of dark red blood per rectum. Bowel movements during the following twelve to twenty-four hours were tarry. The patient had noticed no relationship between these attacks and activity or the taking of food. After experiencing several episodes over a four-week period, he was admitted to his community hospital for diagnosis. There the patient was given only supportive treatment, including three transfusions. During the hospital stay he had a massive gastrointestinal tract hemorrhage and vomited for the first time a small amount of bright red blood. He was discharged from the hosptial on the twelfth day of hospitalization and was referred to the University Hospital for further treatment.

Received for publication, July 26, 1948.

The past history was negative except for a history of "rheumatic pain" between the ages of 6 and 11 years.

Examination at the time of admission to the University Hospital revealed a well-developed and fairly well-nourished white man who showed evidence of marked blood loss. Temperature was 99.8° F., pulse 100, respirations 28, and blood pressure 112/48. The skin was pale, as were the mucous membranes and nail beds. Physical examination otherwise was not remarkable. Abdominal examination was entirely negative.

Laboratory examination at the time of admission revealed a red blood cell count of 2,200,000 with hemoglobin of 7 Gm., and a white blood cell count of 7,000. The blood urea was 36 mg. per 100 c.c. Findings from the blood Wassermann and Kahn were negative. The prothrombin time was 15 seconds as compared with a control of 11.5 seconds. The bleeding time was 2½ minutes and the clotting time 6½ minutes (Lee and White method). The clot retraction was normal. A tourniquet test was negative. Routine urinalysis gave negative results. Stool examination showed a one plus benzidine reaction. The blood type was group O.

A tentative diagnosis of gastrointestinal tract bleeding from duodenal ulcer was made. The patient was placed on an ulcer regime and given a Sippy diet. On the second hospital day an x-ray examination of the gastrointestinal tract was reported to be negative. During the first four days in the hospital the patient was given three transfusions of whole citrated blood, a total of approximately 1,600 c.c., with little change in the hemogram. On the eighth hospital day the patient had a massive hemorrhage from the gastrointestinal tract and passed a large amount of dark red blood with clots. He responded to blood transfusions, and the blood pressure stabilized at 110/60.

The patient was seen in consultation by the surgical service and was tranferred for possible operation.

The patient was placed on nothing by mouth and for the next twelve days was given numerous whole blood transfusions. There was apparently little bleeding from the gastrointestinal tract for several days, and on Sept. 2, 1947, nineteen days after admission, the hemogram revealed 16 Gm. of hemoglobin and 5,600,000 red blood cells. The following day a second gastrointestinal x-ray examination including study of the small bowel was made, which was again reported as negative. Two days later a barium enema was obtained and reported to be negative.

Although all diagnostic procedures had been unrewarding, it was felt that this patient could be bleeding from duodenal ulcer, Meckel's diverticulum, hemangioma of the bowel, or polyps. An exploratory laparotomy was therefore decided upon.

On Sept. 5, 1947, this operation was performed through a right paramedian incision under satisfactory spinal anesthesia. Intra-abdominal examination revealed no abnormalities except for generalized enlargement of the mesenteric lymph nodes, the presence of *Ascaris lumbricoides* in the lumen of the jejunum, and a small, indurated plaque on the surface of the pancreas. On exploratory gastrotomy nothing abnormal was found. The pancreas was biopsied and a mesenteric lymph node and the appendix were removed. The abdomen was closed in layers using interrupted silk sutures. The patient withstood this procedure satisfactorily.

Microscopic study of the tissues removed showed old focal hemorrhage of the pancreas with calcification, mild hyperplasia of lymph nodes, and no pathologic change in the appendix.

The patient's postoperative course was uneventful until the fifth day, when he again suddenly became weak and dizzy. Several minutes later he was discovered by the nurse to be in profound shock. He was given whole blood transfusions immediately and responded well. During the next eighteen days the patient bled frequently from the gastrointestinal tract. The bleeding was of sudden onset, massive in amount, and of short duration. Following each episode of hemorrhage, the blood pressure dropped considerably.

On September 22 the patient had a massive hemorrhage requiring seven whole blood transfusions, a total of 3,760 c.c. Because it was obvious that he was rapidly going downhill and that the prognosis was grave, a second exploratory laparotomy was planned as a last resort.

On September 23 the abdomen was re-opened under endotracheal gas-oxygen-ether anesthesia, through a right paramedian incision. Intra-abdominal examination revealed essentially the same findings as previously. An exploratory duodenotomy in the second portion was carried out and the lumen of the duodenum explored with the gloved finger, which was passed beneath the superior mesenteric vessels into the third portion of the duodenum to the ligament of Treitz. Upon palpating within the lumen over the aorta, a sharp-pointed object was encountered. Immediate re-examination of the x-ray films revealed a shadow over the interspace of the third and fourth lumbar vertebrae that appeared to be a sewing needle. It was lying in the longitudinal axis of the vertebral column. It should be noted that this shadow had been overlooked by all observers in spite of many films on which it appeared.*

Upon incising the ligament of Treitz and reflecting the duodenum upward, a pulsating, tubelike structure about 13 mm. in diameter and 2 cm. in length was found extending from the abdominal aorta to the posterior wall of the duodenum. The duodenum was incised about the attachment of the tube leaving a small cuff of the bowel wall attached to it. The lumen of the bowel was found to be filled with fresh blood clots and bright liquid blood. Upon inspection of the structure from within the lumen of the duodenum, there was seen a small amount of granulation tissue with what appeared to be a small blood clot in the center. During this examination a moderate hemorrhage occurred, and there was a transitory drop in the patient's blood pressure. The pulsating tube was then clamped to prevent further bleeding from the aorta.

Another clamp was applied near its base and interrupted mattress sutures were placed proximal to the clamp. The sac was then excised and another layer of interrupted silk sutures was placed at the aortic end to give added support to the closure. The two incisions in the duodenum were closed transversely, with 00 atraumatic chronic catgut, reinforced by interrupted silk sutures. The needle was lost, presumably in the retroperitoneal tissue, during an attempt to extract it. The abdomen was then closed in layers with interrupted silk sutures.

The patient was placed on continuous duodenal suction for three days following operation. He had an uneventful postoperative course and was up in a chair on the fifth postoperative day. X-ray examination of the abdomen revealed the needle to be still present; however, it was now lying one vertebra lower, anterior to the body of the fifth lumbar vertebra. While in the hospital the patient received a total of 26,500 c.c. of whole citrated blood and 1,200 c.c. of pooled plasma. The patient was discharged on October 2.

He was seen in the surgical outpatient department on Nov. 3, 1947, and again on Jan. 5, 1948, three and one-half months after operation. He was asymptomatic and physical examination was negative. He had gained thirty pounds in weight and had returned to his work in a furniture factory.

The patient was seen again on the afternoon of Feb. 15, 1948, with a history of having passed a stool which contained dark blood following ingestion of his breakfast approximately ten hours previously. He was otherwise asymptomatic. Physical examination at the time of admission was essentially negative except for a questionable bruit in the mid-abdomen in the region of the umbilicus. The blood pressure was 110 mg. Hg systolic and 65 mg. diastolic. Laboratory examination revealed a hemogram of 4,400,000 red blood cells and 13 Gm. of hemoglobin. Rectal examination yielded a glove specimen on which the benzidine reaction was strongly positive.

The patient was admitted to the hospital and placed on nothing by mouth. It was suspected that there was a recurrence of the fistula betwen the aorta and duodenum similar to that found previously.

On the second hospital day the patient was again operated upon under satisfactory endotracheal gas-oxygen-ether anesthesia. The abdomen was opened through the previous wound. A moderate number of adhesions was found beneath the scar and throughout the peritoneal cavity. These adhesions were friable, however, and there was no obstruction of the bowel. Exploration revealed a pulsating mass over the distal portion of the abdominal aorta

*Repeated questioning later failed to elicit any incident suggesting the time and circumstances of its ingestion.

which measured approximately 9 cm. in diameter. Two loops of small intestine were intimately adherent to it. One loop was attached to the anterolateral aspect of the left side of the mass with a fan of its mesentery lying over the surface. The other was adherent to the mass on the right lateral side beneath the mesentery of the other loop. Because of the dense adhesions between the bowel and the mass, it was felt that resection of the two loops was imperative. The more anterior loop of bowel was therefore first resected, leaving a segment approximately 12 cm. in length adherent to the mass. An end-to-end open anastomosis was carried out. The second loop of small bowel was somewhat obscured by scar tissue and adhesions and its relationship to the large bowel could not be readily determined. The loop was clamped on either side of its attachment to the mass, leaving approximately 8 cm. undisturbed. Upon exposing the lumen of this loop, dark blood clots were found. This segment was then determined to be the terminal ileum, which had been divided approximately at the ileo-cecal valve. Ileocecostomy re-establishing the normal relationships was therefore performed.

The posterior peritoneum was incised in the midline and reflected from the mass laterally. Through the upper end of the incised peritoneum, the aorta was isolated above the mass and a Shenstone tourniquet carrying a rubber tube was placed. Similar procedures were done with both common iliac arteries. With the tourniquets tightened so as to occlude the lumina of the arteries, the false aneurysm carrying the two resected loops was dissected from the aorta. A small opening into its lumen was identified, measuring approximately 1½ cm. in length. Interrupted 000 braided silk sutures were placed in the wall of the aorta to close the defect. While the first suture was being placed, the end of the needle was identified and withdrawn from the wall of the aorta. It was markedly oxidized. The surface was black and rough. The eye was recognizable and the other end was roughly pointed so that for the moment complete removal was believed to have been accomplished. Following closure of the opening in the aorta with one layer of interrupted silk sutures, the tourniquets were released from about the vessels beginning with those on the common iliac arteries. No bleeding followed. The aorta was occluded completely for approximately ten to twelve minutes. A small segment of fascia from the anterior rectus sheath was excised and this patch was sutured over the suture line in the aortic wall. Border segments of the false aneurysm that had not been removed were overlapped by suture so as to cover the fascial patch. The posterior peritoneum was closed with a continuous suture of atraumatic catgut after placing 100,000 units of penicillin and ½ Gm. of streptomycin in the retroperitoneal space because of the contamination from opening the intestine. One hundred thousand units of penicillin and ½ Gm. streptomycin were also instilled into the peritoneal cavity and the abdomen was closed without drainage. While on the operating table the patient received 2,000 c.c. of whole citrated blood.

Microscopic sections of the bowel and aneurysmal sac showed acute and chronic inflammation of the small intestine with scarring and foreign body reaction and partially organized blood clot.

Postoperatively the patient had an uneventful course. Arterial pulsations in the lower extremities were normal and there was no muscular weakness. He began taking a diet on the sixth postoperative day and was allowed out of bed. A postoperative roentgenogram revealed a shadow about 0.5 cm. in length, presumably representing a fragment of the needle that had spontaneously separated through oxidation and had remained in the abdomen, possibly within the lumen of the aorta.

The patient was discharged on the twelfth postoperative day. On March 19, 1948, and again on April 19, 1948, he was seen in the outpatient department. He was asymptomatic and physical examination was negative.

The patient's latest admission to the hospital was on May 14, 1948, approximately three months after the last discharge. He had been symptom-free but had been nervous and greatly concerned about his health. On the morning of the day of the last admission he passed a tarry stool. There had been no weakness, sweating, or pain on this occasion.

Physical examination revealed a well-developed and nourished young man appearing somewhat disturbed. The blood pressure was 122 mg. Hg systolic and 80 mg. diastolic, pulse 84, respirations 18. Abdominal examination was negative. A glove specimen from the rectum yielded a positive benzidine reaction. Laboratory examination revealed hemoglobin of 13 Gm., 4,030,000 red cells, and 7,600 white cells. The urinalysis was negative. An x-ray view of the abdomen showed no change in the position of the foreign body since the last examination.

The day following admission to the hospital a gastrointestinal x-ray series and small bowel study were obtained but showed no abnormality. The history and findings at the previous operations suggested the wisdom of prompt exploration.

An exploratory laparotomy was performed under gas-oxygen-ether anesthesia. Minimal adhesions were found. Exploration revealed no adhesions between the small bowel and the posterior peritoneum. The entire abdominal aorta and the great vessels were palpated under direct vision and found to be normal. The third portion of the duodenum was not adherent as it had been at the first operation. An exploratory gastrotomy was again done because of some edema about the pylorus. No pathologic change was found. The small bowel was transilluminated throughout its length and no abnormal shadows were seen. The abdomen was closed in layers with silk.

The patient had an uneventful postoperative course and was discharged from the hospital on the seventh postoperative day. He has remained asymptomatic with reassurance and an ulcer regime. At the end of eight weeks he had gained twelve pounds in weight and was working.

## COMMENT

This report was first prepared prior to the last episode of hemorrhage. The relationship between the swallowed foreign body and the two aorto-intestinal fistulas had been established by the identification of the needle at the site of each fistula. The most recent episode of hemorrhage into the bowel is unexplained, although it was proved that it was not due to a fistula between the bowel and any of the great vessels. Although no duodenal ulcer was found by x-ray or on exploration, the patient was treated by an ulcer regime, since his psychic make-up, his recent repeated severe illnesses and surgical operations, and his obvious apprehension made this diagnosis a possibility. The cause of the last episode of bleeding must remain obscure up to now. It should be noted that it was a single hemorrhage minor in degree and has not been repeated as the earlier hemorrhages were. It should also be noted that a fragment of the needle still remains in the aortic lumen, its wall, or the retroperitoneal tissues.

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# Case Reports

## PERFORATION OF THE COLON FOLLOWING THE USE OF ENEMAS

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IT HAS been our unfortunate experience to have three fatal cases of perforation of the colon precipitated by enemas in a period of three months. The diagnosis was confirmed by autopsy in one case and at operation in the other two cases.

Enemas have been used almost universally since 1000 to 2000 B.C., according to Lieberman,[1] for disorders of the large bowel. In recent years, however, the efficacy of enemas or colonic irrigations has been disputed in many disorders of the large bowel, especially their habitual or repeated use. Hibben[2] said that the present indications for enemas are constipation, fecal impaction, amebiasis, diverticulosis, and mucous colitis. In addition to some of these, most clinics use enemas preparatory to proctosigmoidoscopy and barium enema. We do not wish to be controversial regarding the indications for enemas; however, we feel that the condition of the large bowel, as determined by proctosigmoidocopy, barium enema, and stool examination, should be determined before any plan of therapy is instituted.

By far, the most important aspects in the administration of enemas are the liquid used, the amount of liquid, and the force used to inject it. There have been many reports by Barker[3] of acute colitis resulting from the use of soapsuds enemas, and we too have noticed that a mild proctocolitis sometimes followed this type of enema when used preparatory to proctosigmoidoscopy. Tap water or normal saline enemas are more physiologic and produce the desired effects in our experience. In regard to the amount, we have found that one to two pints is usually sufficient to cleanse the distal colon or to induce defecation. Last, and most important, is the force used to inject the enema; here we feel that the enema can should never be placed higher than two and one-half feet above the bed and that the liquid should be given slowly in an interrupted stream. To impress this upon the nursing and resident staff we are taking small groups to the fluoroscopy room to observe barium enemas and their effects on the bowel.

In the three cases that we are presenting, the perforation was proximal to an annular carcinoma of the sigmoid colon—below the promontory of the sacrum in one case, and proximal to a carcinoma of the rectosigmoid in two cases. We feel that the enemas were definitely responsible for the immediate deaths in these patients because all three perforations were proximal to the lesions in the colon, also because the patients experienced a sudden shock in the lower abdomen immediately following the administration of the enemas, and of course because of the operative and autopsy findings. As Wangensteen[4] pointed out, high obstructions are no longer the most serious to life, for low obstructions hold a far

Received for publication, Aug. 20, 1948.

greater risk due to sustained intraluminal pressures that threaten the viability of the bowel wall. When we add to this an enema forced past an almost complete obstruction it is no wonder that perforation occurs so easily. As can be seen while doing a barium enema, the sudden forceful injection of the barium irritates the colon and initiates a massive peristaltic movement which can eject the enema with great velocity and force. It is by this mechanism, we feel, along with a weakened bowel wall due to prolonged distention and malignant change that the perforations occurred. Pratt and Jackman[5] have reported perforations of the rectal wall by enema tips. This can be ruled out in our cases since the perforation sites were proximal to the obstructing lesions and inaccessible to trauma from an enema tip.

## CASE REPORTS

CASE 1.—W. P., a 77-year-old white man, was admitted to the York Hospital on Nov. 9, 1947, complaining of pain in the abdomen, nausea, and vomiting.

The patient had a slowly progressive constipation of two years' duration. The week prior to admission he had been seized with lower abdominal pain; he consulted his family physician who advised x-ray studies of the patient and an enema. The enema gave relief for several days; however, the day prior to admission he again experienced the lower abdominal pain accompanied by some distention of the abdomen. An enema was again advised and when the pain returned the enema was repeated, following which the patient said he felt something "pop" in the lower abdomen. He immediately became nauseated, cold, began perspiring, and was sent to the hospital.

On physical examination the patient was found to be an emaciated, elderly white man in acute abdominal distress and in a mild state of shock. Blood pressure was 90/60, pulse 120, and temperature 96.4° F. He had an enlarged heart with loud snapping sounds and a loud systolic murmur heard best over the apex. The abdomen was boardlike with no difference of sensation in any area. A large polypoid mass could be palpated rectally at the tip of the finger on the anterior rectal wall.

Impression was a generalized peritonitis secondary to traumatic injury of the lower sigmoid, probably due to the enema, and carcinoma of the rectosigmoid.

The patient was treated with intravenous fluids and sedation and because of his moribund condition, therapy was limited to this. He died six hours following admission.

At autopsy a generalized peritonitis with a watery fecal contamination of the abdomen was found. There was a 3 cm. perforation of the bowel just proximal to the rectosigmoid which was the seat of a carcinoma of the rectum. There was no evidence of metastases to the liver. However, there were several nodes palpable along the abdominal aorta which were grossly involved.

CASE 2.—F. B., an 80-year-old white man, was admitted to the York Hospital on Dec. 9, 1947, complaining of severe abdominal pain.

The patient had been well until one month prior to admission, when he began to experience weakness, anorexia, and a vague abdominal discomfort. One week prior to admission he began to notice that stools were becoming scant and contained some blood. The day of admission, immediately following an enema, he experienced a sharp pain in the lower abdomen; a physician was summoned and hospitalization was advised.

On examination temperature was 99° F., pulse 76, respirations were 18, and blood pressure was 140/80. The patient was poorly nourished, pale, dehydrated, and complaining of lower abdominal pain. Heart rate was normal but the rhythm was irregular, with systolic murmurs heard over the mitral and aortic areas. The lungs were clear to auscultation and percussion. The abdomen was flat and rigid with generalized tenderness which was more marked in the lower abdomen. There were bilateral reducible inguinal hernias.

On rectal examination a firm, fixed, and tender mass could be palpated by the tip of the finger.

Impression was generalized peritonitis secondary to perforation of the lower sigmoid and carcinoma of the rectosigmoid.

Laboratory studies revealed urinalysis, trace of albumin, otherwise normal; 10.5 Gm. of hemoglobin; 3,790,000 red blood cells; 8,350 white blood cells, 85 per cent neutrophiles; 15 per cent small lymphocytes. Kahn and Wassermann were negative. Blood urea nitrogen was 39.4 mg. Abdominal culture showed usual large bowel flora.

Operation was performed on Dec. 10, 1947, under open-drop ether anesthesia. At this time a perforation of the sigmoid, 3 cm. proximal to the rectosigmoid junction, was found and distal to this an annular carcinoma which had almost completely obstructed the bowel. There was no evidence of metastases in the liver or regional nodes. There was a generalized peritonitis with scybala floating in the abdominal cavity. A loop colostomy of the sigmoid was performed and the abdomen aspirated as well as possible.

The patient was treated with large doses of streptomycin and penicillin, along with intravenous fluids and several blood transfusions. The patient seemed to improve until the third day postoperatively when the heart became irregular and rapid. He gradually became more toxic in spite of rapid digitalization and died on Dec. 12, 1947, three days after admission.

Case 3.—L. J., a 59-year-old white woman, was admitted to the York Hospital on Jan. 30, 1948, with the chief complaint of pain in the abdomen.

For the past year the patient had been having progressive constipation and occasional bleeding upon defecation. The patient treated herself with cathartics until two weeks prior to admission, when she could no longer obtain relief. Her family physician was summoned and advised an enema and x-ray studies of the colon. Following the enema she felt something "pop" in the lower abdomen; there was distention and pain in the entire abdomen shortly thereafter. Hospitalization was then advised.

On admission the patient was found to be a very obese white woman in acute abdominal distress from pain and distention of the abdomen. Temperature was 98° F., pulse 100, and blood pressure 120/82. Cardiovascular and respiratory systems were normal on examination, but the abdomen was markedly distended, rigid, and tender generally. Borborygmi and peristalsis were present. Rectal examination was normal except for bright red blood on the glove when the finger was withdrawn.

Impression was generalized peritonitis secondary to perforation of the colon and carcinoma of the sigmoid colon or descending colon.

Laboratory studies showed urinalysis normal; blood, 14.8 Gm. of hemoglobin; 4,790,000 red blood cells; 11,240 white blood cells; 89 per cent neutrophiles; 6 per cent small lymphocytes, and 5 per cent monocytes. Kahn reaction was negative. Nonprotein nitrogen was 51 mg. Blood proteins were 7.7 Gm. Albumin-globulin ratio was 1.5 to 1. Barium enema revealed complete obstruction of the lower sigmoid with marked distention of the large and small bowel proximal to this.

A Miller-Abbott tube was passed and intravenous fluids were started along with penicillin and sulfadiazine therapy.

Operation was performed on Feb. 1, 1948, at which time about 1000 c.c. of brownish-red, foul-smelling fluid rushed from the abdomen. In the pelvis was found a carcinoma of the sigmoid with a perforation along the lateral side and proximal to the carcinoma. The abdomen was aspirated as thoroughly as possible and the perforation closed. A Witzel colostomy was performed because the obesity of the patient would not allow us to form a loop colostomy of the sigmoid. The Miller-Abbott tube was found in the proximal ileum, functioning normally. There was no evidence of liver metastases; however, involved nodes were palpable in the sigmoid mesocolon.

The patient became more toxic; respirations became more labored and finally ceased two days after operation.

From these cases it can be seen that therapy in the form of enemas was instituted before a thorough examination of the rectum, colon, and feces was performed, a practice which should be terminated especially in middle-aged and elderly patients. The use of the 3-H enema (high, hot and a h____ of a lot) is to be condemned and was the method employed in two of our three cases. It is the direct responsibility of the physician to determine when an enema should be given, how often an enema should be given, and to instruct the person administering the enema in the exact technique he should follow.

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# FIBROMA OF THE STOMACH

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AMONG benign neoplasms of the stomach, pure fibromas are rare. According to our survey less than 125 cases have been reported to date. The tabulation of Dudley, Miscall, and Morse[1] accounts for 97 of these cases. Most of the remaining ones are individual case reports. The size of the growths varied considerably. The largest one was reported by Scharapo.[2] It was surgically removed from a 24-year-old woman and weighed 5,500 Gm. The next largest, weighing 3,660 Gm., was reported by Pendl.[3] This was discovered at necropsy of an 81-year-old man. In this paper a large fibroma of the stomach is reported which was surgically removed from a 17-year-old Negro girl. This is believed to be the second largest such growth on record.

## REPORT OF CASE

A 17-year-old Negro girl was admitted to the University of Oklahoma Hospitals, March 11, 1947, complaining of a mass in the abdomen and shortness of breath on exertion. When she was first seen in the outpatient department, Feb. 25, 1947, a large, smooth, nontender mass was palpable in the upper abdomen. Roentgenograms of the chest were negative; those of the abdomen disclosed that the mass displaced the transverse colon downward and the stomach upward and anteriorly (Figs. 1 and 2). Intravenous urograms revealed some distortion of the lower calyx of the left kidney attributed to pressure from the mass. Pelvic examination revealed slight enlargement of the cervix uteri, a small erosion of the os, and a moderate mucoid discharge. The mass seemed not to be connected with either the uterus or the adnexa.

At the time of admission, physical examination yielded no additional information. Examination of the blood and urinalysis gave negative results except for a 1 to 2+ albumin. On cystoscopic examination there was some injection of the bladder mucosa.

Laparotomy March 28 disclosed a smooth, firm, well-circumscribed neoplastic mass about 20 cm. in diameter attached to the greater curvature of the stomach and to a portion of the transverse colon and situated apparently within the omental bursa. The mass was removed together with a portion of the stomach and a segment of the transverse colon. The opening in the stomach was repaired, and the severed portions of the transverse colon were sutured to the abdominal wall by a "double barrel" colostomy. A jejunostomy was then made utilizing a portion 12 cm. past the duodenojejunal junction. The patient recovered slowly under supportive therapy. The jejunal tube was removed on the fourteenth day. The opening closed spontaneously a few days later. The colostomy was functioning well when the patient was discharged May 2. She was readmitted June 28, and on July 17 the colostomy was closed. The patient made an uneventful recovery and was discharged on July 30. She was last seen in the outpatient department March 6, 1948. At this time she had gained about twenty-five pounds and was asymptomatic, except for a sense of fullness before finishing meals. Roentgenograms disclosed some narrowing of the transverse colon near the splenic flexure with no hindrance to the passage of content. There was some gastric residuum three hours after a barium meal.

The specimen obtained at operation consisted of a firm, encapsulated mass 25 by 19 by 16 cm., weighing 4,315 Gm. (Fig. 3). Blood vessels were prominent beneath the capsule

Received for publication, June 21, 1948.

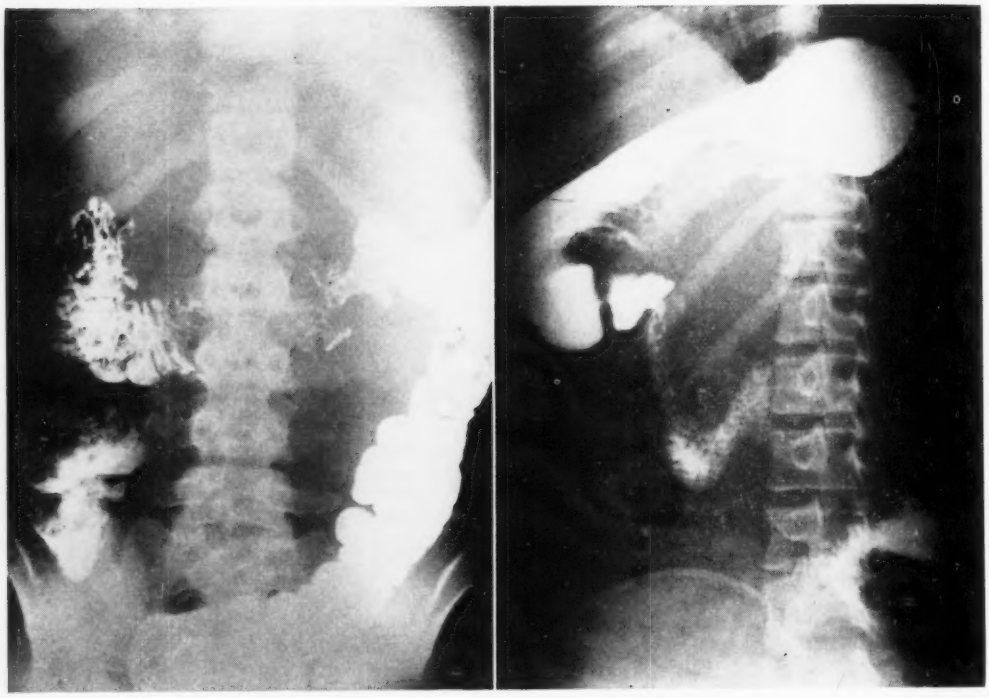

Fig. 1. Fig. 2.

Figs. 1 and 2.—Roentgenologic appearance of the growth. The mass displaced the transverse colon downward, and the stomach upward and anteriorly.

Fig. 3.—The appearance of the growth externally and on the cut surface. The resected portion of stomach is seen on the upper surface.

and the serosal covering. Over an area 11 by 4 cm. and closely adherent to the surface was a portion of stomach. The pattern of the gastric mucosa overlying the attached portion of the stomach was clearly discernible with no distortion or discoloration. A segment of large intestine 16 cm. long was adherent to another portion of the mass. The cut surfaces of the mass bulged slightly and were gray with a streaked pattern of interlacing fibers. There were no areas of softening, necrosis, or hemorrhage. The pattern was identical on several of the cut surfaces.

Fig. 4.

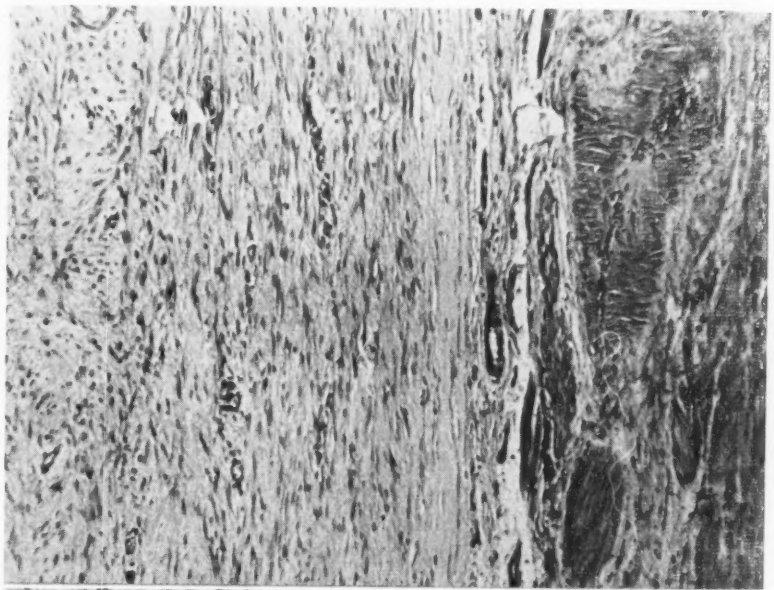

Fig. 5.

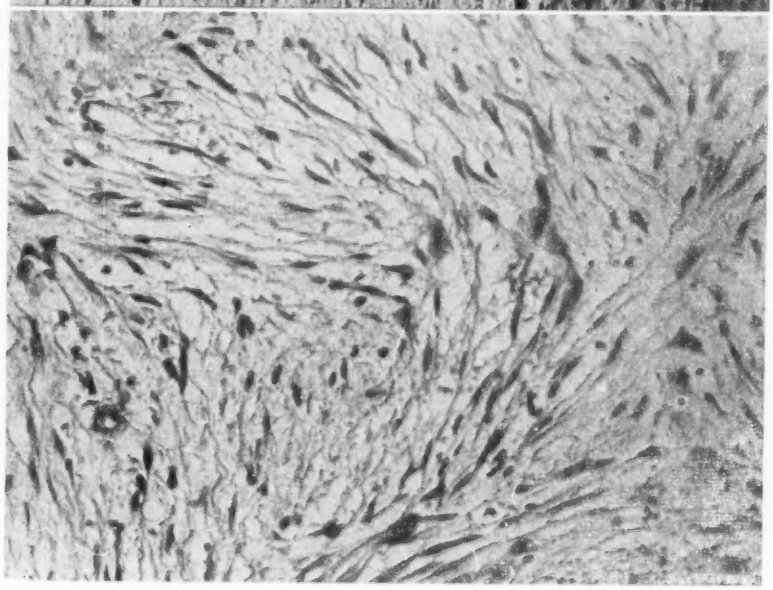

Fig. 4.—The growth appears to originate from the perimuscular connective tissue of the stomach with an almost straight line of demarcation (×100).

Fig. 5.—Microscopic appearance of the growth (×300).

Microscopic preparations stained with hematoxylin and eosin and representing many parts of the growth disclosed spindle-shaped cell nuclei in streams and whorls within a delicate, fibrillar ground substance (Figs. 4 and 5). In some fields the nuclei were large and vesicular with stellate cytoplasmic processes fading into the fibrillar ground substance. Coarse collagenous bundles were seen here and there. There were no areas of hemorrhage or necrosis. The growth seemed to originate from the perimuscular connective tissue of the wall of the stomach with an almost straight line of demarcation. No changes were noted in the muscular coats, submucosa, or mucosa of the stomach overlying the growth.

## COMMENT

The appearance of the growth and its gross and microscopic relations to the stomach strongly suggest that it arose from the connective tissue between the muscular coat and the serosa as in the case reported by Pendl.[3] The benign nature of the growth is evidenced by its microscopic structure and noninvasive character. Since the growth remained well circumscribed, complete removal was feasible and a cure could thus be effected.

## SUMMARY

Fibroma of the stomach was successfully removed from a 17-year-old Negro girl. This is believed to be second largest such growth on record.

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# *Surgical Technique*

## PECTORAL MYOPLASTY FOR CLOSURE OF RESIDUAL EMPYEMA CAVITY AND BRONCHIAL FISTULA

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THE pectoralis major muscle meets the anatomic requirements for a successful myoplasty. It is useful in completing the obliteration of apical chronic empyema cavities in conjunction with thoracoplasty. Such a procedure may be employed when decortication is not feasible or when a chronic empyema follows pulmonary resection. This type of myoplasty may also be utilized to close a bronchial fistula following lobectomy of the upper lobe or pneumonectomy. The pectoral muscle may occasionally be employed as a "fill" for a residual apical intrapulmonary cavity without epithelial lining when pulmonary resection is contraindicated. Of the various muscles which we have employed during the past ten years for plastic closure of intrathoracic defects, the pectoralis major has proved to be the most satisfactory. In some cases the pectoralis minor muscle was also employed. Pectoral myoplasty has been performed sporadically in the past but the technique then employed often led to unsatisfactory results. The method which we are describing was originally suggested to us by Dr. Frank Berry. The procedure has now been employed in over twenty patients with good results.

Certain fundamental principles must be observed in the utilization of the pectoral muscles for obliteration of a residual pleural or intrapulmonary cavity. The cavity which is to receive the muscle graft must be relatively clean. Failure of healing with loss of muscle may follow an attempt to place the muscle in an unprepared cavity. It is usually best to perform the myoplasty at a second operation, the cavity having been drained or unroofed at the time of the first procedure. Unhealthy granulation tissue in an empyema cavity should be removed with gauze or curette. All recesses of an empyema cavity should be explored and laid open in order that no sinus leading from the main part of the cavity may later cause a localized abscess. Antibiotics are administered parenterally in the preparation of the patient for operation.

That portion of the pectoralis major muscle which has its origin from the sternum and chest wall is freed by blunt dissection in the axillary region (Fig. 1). The upper fibers which extend from the clavicle to the humerus are not included. The attachment of the freed portion of the muscle to the humerus is severed. The origin of the muscle from the chest wall is not disturbed. If the

Received for publication, Dec. 10, 1948.

pectoralis minor is also employed in order to obtain a little additional muscle, the insertion on the coracoid process is the end which is usually mobilized, but in some cases it may be best to sever the costal attachment and use that end. Occasionally the pectoralis minor may be utilized to fill the narrow apical portion of an empyema cavity or to suture over a bronchial fistula while the pectoralis major is placed in the main part of the empyema cavity.

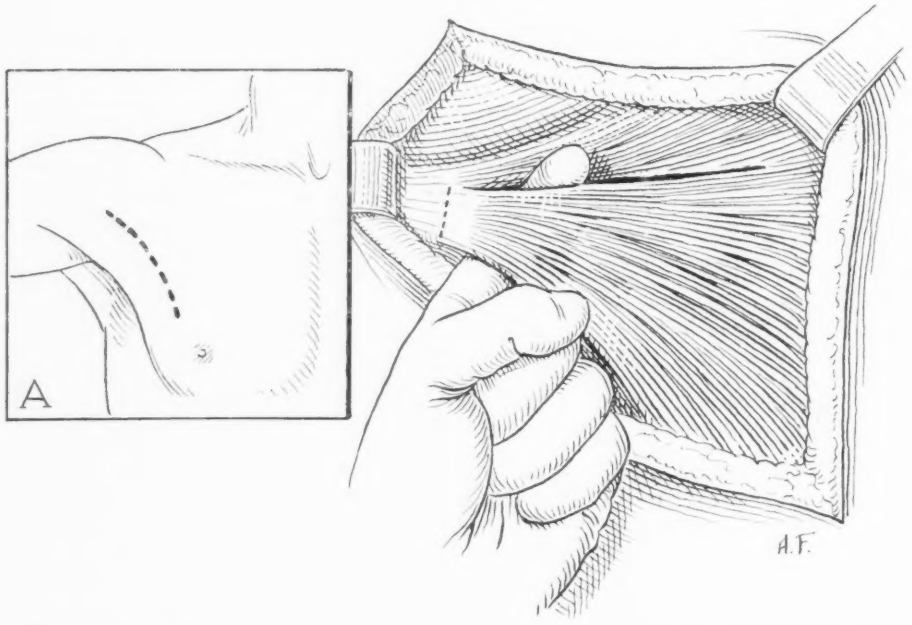


Fig. 1.—The axillary portion of the pectoralis major muscle is exposed. That part of the muscle which is to be employed for the myoplasty has been bluntly separated from the clavicular fibers. The broken line indicates the site of section of these muscle bundles close to their humeral attachment.

*A*, Insert shows usual skin incision which may be modified because of previous incisions. Scars which cause axillary contraction should be avoided.

Mobilization of the muscle employed for the graft should involve minimal disturbance of the nerve supply. It is essential that the blood supply be adequate. When the muscle is inserted into the space which is to be obliterated, the muscle should be in contact with the inner wall of the cavity; tenting of the muscle across the upper part of the cavity is to be avoided. Splitting of the pectoralis major along the course of the muscle bundles into two or occasionally even three components may permit better placement within the cavity. When a relatively large empyema cavity is present, it is preferable to fill one part of the cavity adequately rather than to spread the muscle over too large an area. The sutures which are employed to maintain the muscle in proper position should be so placed as to minimize interference with the blood supply to the muscle and to avoid tenting and tension (Fig. 2). The local use of penicillin or streptomycin is frequently indicated. Drains should be so placed that they do not interefere with adherence of the muscle to the walls of the cavity. Hematomas and collection of serum under the flap should be avoided as much as

possible. It is often best to close the outer part of the wound relatively loosely to permit a free escape of blood and serum. The application of a firm pressure dressing is an essential part of the procedure. The pressure dressing is sometimes more helpful than the sutures in maintaining the muscle in proper position during the healing period. We prefer not to remove the original dressing for seven to ten days even though profuse drainage may occur and in spite of the fact that the dressing will sometimes become quite odorous. If the drain is to be removed before the first change of dressing, heavy silk sutures should be attached to the drain and brought out through a corner of the wound so that it can be extracted without complete change of dressing. Adequate doses of antibiotics in the postoperative period are most important since the operative procedure is usually performed in a grossly contaminated field.

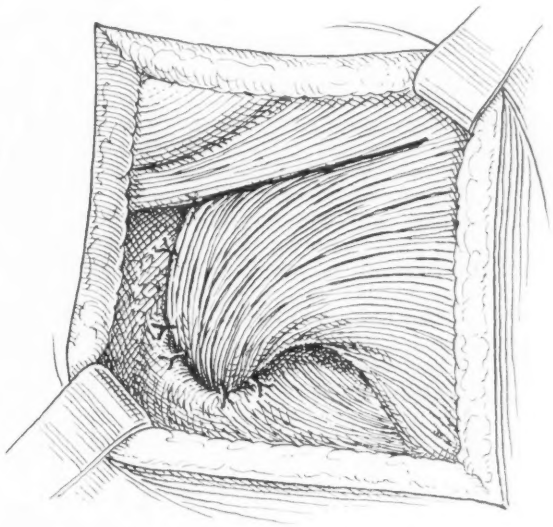

Fig. 2.—The mobilized axillary portion of the pectoralis major muscle has been turned backward and inward. Several interrupted sutures have been inserted to hold the muscle in place.

Some dissatisfaction with pectoral myoplasty expressed in the literature is due to mobilization of the origin rather than the insertion of the muscle. Because of its contour, the portion of the pectoralis major which is attached to the chest wall is quite unsatisfactory for a myoplasty. Moreover, when the muscle is left attached to the humerus, motion of the arm will tend to displace the graft. There are numerous advantages from an anatomic standpoint in dividing the insertion rather than the origin of the pectoralis major muscle when employing it for a pedicle muscle graft. The blood supply from the thoracoacromial and internal mammary vessels is preserved. The nerve supply from the medial and lateral anterior thoracic nerves is not interrupted. The pectoralis major, being a bilaminar muscle, can be unfolded and thus fill a large, shallow cavity. If a smaller, deeper cavity is to be filled, the muscle is best used in its original form. The humeral end of the pectoralis major contains some tendinous parts which can be utilized for anchoring sutures. However, since muscle fibers constitute the major portion of the humeral end, the apex of the

cavity is filled mainly with vascular muscle tissue rather than avascular tendinous strands. Motion of the arm can be resumed immediately following operation since traction on the graft will not occur. No interference with arm function results from the detachment of the insertion of the pectoral muscles even though the trapezius, rhomboids, and latissimus dorsi have been divided during the performance of a thoracoplasty at previous operations through a posterior parascapular incision.

The pectoral muscle has been employed particularly to complete the obliteration of tuberculous empyema cavities. In most instances an extrapleural or Schede thoracoplasty had already been performed at previous operations. When the residual empyema cavity is in the midaxillary or upper anterior thoracic region, the pectoral muscle is ideally located for grafting. If the empyema cavity is located posteriorly, the pectoralis major can still be employed provided that the anteroposterior distance has been reduced by an extensive thoracoplasty which has included removal of the anterior ends of the upper ribs. If the anterior rib segments remain, the pectoral muscle may not reach the empyema cavity because of the increased distance which must then be traversed.

When the pectoralis major muscle is employed to aid in the obliteration of an intrapulmonary cavity, usually only a portion of the lower border of muscle is required. That amount of muscle which is needed is freed from its humeral attachment and split away from the remainder of the muscle.

## SUMMARY

The pectoralis major provides a very satisfactory muscle pedicle for plastic obliteration of residual empyema cavities when the insertion is detached from the humerus. The muscle may also be employed for closure of some bronchial fistulas. The method advocated permits good mobilization of the pectoralis major muscle without interference of the blood or nerve supply. This type of myoplasty has been employed in more than twenty patients with good results.

# Editorial

## Some Aspects of the Problem of Cancer Research

THE clamor about cancer, steadily increasing as it is, disturbs the conservative physician, pleases the investigator, and confuses the public. Two grounds for it are apparent: cancer is dreaded and feared by all, while at the same time it represents a peculiar challenge to the medical scientist. A more important reason for emphasis on the problem, although one often overlooked, is the fact that substantial progress toward the more adequate control of cancer in human beings is steadily being made.

Popular interest in cancer study has been focused principally on the programs in fundamental research which are now under way on an extensive national scale. These provide particularly convincing evidence of the increasing public realization of the value of investigation to medicine and of a general willingness to contribute to its support.

Important as basic studies are, however, and as clearly as their results indicate revolutionary discoveries to come, they obscure somewhat the extraordinary progress which is being made today in the practical control of cancer in man. In no other field of chronic disease is advance so rapid, and in no other is the breach of misunderstanding so great between the workers in fundamental and those engaged in applied studies.

The care of patients with cancer is viewed, quite properly, as a function of the medically trained man. Some see it, indeed, as the exclusive property of the surgeon. But it is not and clearly cannot be the sole area of any one discipline, because of the rapidly increasing complexity of medical procedure. Accurate and early diagnosis depends upon the competence of the cytologist, the physicist, and the educator quite as much as it does on that of the practitioner. Curative treament, still primarily surgical, is undergoing a major metamorphosis, in small part only because of better handicraft and to a greater extent because of the work of the bacteriologist, mycologist, chemist, and physiologist.

The study of cancer, conventionally done in lower forms in contrast to its clinical care and study in patients, is considered, correctly, to be a problem in tissue and cellular growth, both normal and abnormal. Unfortunately, in this field, the fundamental scientist feels that he has sole rights. As a result of this concept he views practically every branch of natural science as pertaining to the cancer problem. Then, since substantial funds are available for cancer research, every scientist contends vigorously that his study is a proper and deserving financial beneficiary of them. This would not be troublesome were there enough funds to go around, but this is out of the question since general scientific programs can absorb almost any amount of

money. Hence, it becomes very difficult under existing circumstances to sandwich in the work which should and does bring the advances of basic research to bear on the problems of clinical cancer. It is assumed, mistakenly, that the concurrent existence of new basic knowledge on the one hand, and of desperate problems of disease on the other, will automatically, by some sort of spontaneous combustion, generate new and applicable discoveries. Although this is certain to occur eventually, too often it is long deferred unless vigorously catalyzed. Delay cannot be tolerated in the solution of major problems of public health.

It becomes, then, the function of medicine to examine the facts carefully and to decide on the basis of detailed study what will lead most rapidly to further progress. If this is done concerning the cancer problem, an extremely interesting development is to be seen. When adjusted for the average age of the population, the death rate is dropping perceptibly for cancer in general and dramatically for cancer of certain sites.* This is perceived, however, only if the figures are corrected, since the trend is masked by the upturn in the total number of deaths from cancer due to the steadily and rapidly increasing age of the individuals who die. One exception to this general rule is at hand in cancer of the lung.

Since real progress is being made, a knowledge of the factors responsible for it should provide a reliable basis for the planning of future work in such a way as to permit even more rapid and impressive advance. It is clear that the gains made so far are not due to a better understanding of the fundamental cause of cancer in human beings. This is essentially as obscure today as it was two centuries ago. Headway has been made because of two simple basic concepts developed through long and bitter experience. These are that the only good cancer is one which has been wholly extirpated, and that the ability to remove it completely increases rapidly with early diagnosis. These are not very complex principles. They appear, indeed, to be obvious, but it is astonishing how frequently they are overlooked as bases of policy. Late diagnosis and incomplete surgical procedures are still the greatest friends of death from neoplastic disease.

Earlier recognition is being accomplished through public and professional education and the development of new diagnostic procedures. From the results of one study† it appears that delay is one-half as frequent today as it was eight years ago. Treatment is being instituted very much earlier for some sites, notably breast and uterus.

Cure rates as a result of surgery have increased substantially for many forms of cancer. Surgical technique improves constantly, but more important still, fundamental knowledge derived from fields apparently wholly unrelated to cancer is expanding enormously the scope of effective treatment. Total laryngectomy is an old procedure formerly attended by prohibitive mor-

*Encouraging Trends in Cancer Mortality, Statist. Bull. Metrop. Life Insur. Co. **26**: 1-3, 1945.

†Leach, J. E., and Robbins, G. F.: Delay in the Diagnosis of Cancer, J. A. M. A. **135**: 5-8, 1947.

tality; now it is routine and without significant hazard if in good hands. Total cystectomy with ureterocolic anastomosis for bladder cancer is now standard in some clinics and should yield cure rates of over 40 per cent, in contrast to the unsatisfactory figure of 6 per cent obtained by irradiation or less complete procedures. Radical surgery for cancer of the pelvic viscera is now justified by its results, whatever the opposition to it may be. These procedures are possible because of the application to cancer surgery of advances in physiology, biochemistry, pathology, and bacteriology.

Perhaps it would be well for those charged with responsibility for the decisions regarding the support of cancer research to realize that the mortality from the disease is real and very substantial. Many of these patients die with cancer wholly localized and susceptible to surgical removal by radical procedures. This is particularly apparent for cancer of the bladder as shown by the studies of Jewett,* and for uterine neoplasms from the observations of Bonney.† The cure or substantial palliation of these patients by extensive operative techniques is a complex matter involving the most adequate application of the best methods of modern physiologic science, but it can be done. Much room for improvement and further lowering of operative mortality remain. These will come, for the present, from the work of the laboratory closely integrated with the treatment of cancer in man. Here is an area of investigation, not adequately financed or staffed at the present time, which is certain to achieve substantial reward in terms of lives saved. It merits as serious consideration and support, as much dignity and stature as the important but supposedly more basic studies. Neither scientific area is exclusive of the other; they are mutually complementary and, if so conducted, important advance is certain to result, measurable factually in terms of lives saved.

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New York, N. Y.

*Jewett, H. J.: Infiltrating Carcinoma of the Bladder; Application of Pathologic Observations to Clinical Diagnosis and Prognosis, J. A. M. A. **134**: 496-500, 1947.

†Bonney, V.: The Results of 500 Cases of Wertheim's Operation for Carcinoma of the Cervix, J. Obst. & Gynaec. Brit. Emp. **48**: 421-435, 1941.

# Recent Advances in Surgery

Conducted by Alfred Blalock, M.D.

## THE SURGICAL SIGNIFICANCE OF DISSECTING AORTIC ANEURYSMS

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*(From the Department of Surgery, the Alexander Blain Hospital)*

DISSECTING aneurysm is a disease produced by the dissection of the wall of a large vessel, usually the aorta, by blood. The intima and inner portion of the media are separated from the outer media and adventitia. This lesion ordinarily occurs as an acute process and gives rise to diffuse and protean clinical manifestations. The pathogenesis of this lesion has been discussed previously[1] and need not be considered here.

Pathologists have recognized dissecting aneurysm since Laennec gave the first description of this disease. In recent years, internists, and especially cardiologists, have become more aware of its existence. Indeed, now some 30 per cent of dissecting aneurysms are diagnosed ante mortem.

Because we have seen this disease produce symptoms which appeared surgical in nature, in our own experience, we believed it worth while to review the recent literature in an attempt to determine the incidence of mistaken surgical diagnoses made in this primarily nonsurgical disorder.

As the following study shows, the time has arrived when surgeons, too, must be familiar with this entity in order that unnecessary and futile operations may be avoided.

### METHOD

All the cases of dissecting aortic aneurysms, in the American literature from 1936 through 1947, reported in sufficient detail to include a differential diagnosis were analyzed. Those diagnosed as surgical diseases and those in which the diagnosis of surgical disease was considered in the differential diagnosis were studied in detail. The leading symptoms, ante-mortem and post-mortem diagnosis, treatment, if any, and the extent of the dissection were noted.

### ANALYSIS OF CASES

In the past eleven years, there have been 278 cases of dissecting aneurysms reported in detail sufficient for study. Thirty-six cases, or 12.9 per cent, were confused with or diagnosed as surgical disease. There were innumerable cases reported where surgical disease must have been considered because of the signs, symptoms, laboratory work, and x-ray findings, but since there was no mention of surgical disease in the differential diagnosis, these cases were not included in our study. Twenty-one of these thirty-six cases, or 7.6 per cent of the total number of cases, were diagnosed as surgical diseases. In the remaining fifteen

Presented in part at the Second Annual Meeting of the American Society for the Study of Arteriosclerosis, Chicago, Ill., Nov. 3, 1947.

cases, 5.6 per cent of the total number, surgical disease was considered in the differential diagnosis. Of the twenty-one cases diagnosed as surgical emergencies, eleven patients, or 52 per cent, underwent unnecessary operative procedures, either for diagnostic purposes or for treatment of the disease diagnosed. In one patient (Case 12) in whom surgical disease was considered in the differential diagnosis, a surgeon would have performed an early but useless operation if the internist had not insisted on the correct diagnosis.

The diseases considered in the differential diagnosis or mistaken for a dissecting aneurysm included (Tables I and II): Embolus of iliac artery, thrombus of femoral artery, acute surgical abdomen, kidney tumor or nephrolithiasis, pulmonary or mediastinal tumor, pancreatic or mesenteric cyst, subdiaphragmatic abscess, and embolus of the brachial artery.

The operations performed (Table I) included embolectomy, lumbar sympathetic block, abdominal exploratory laparotomy, and bronchoscopy.

TABLE I. SURGICAL DISEASES MISTAKEN FOR DISSECTING ANEURYSM

| DISEASES | | OPERATIONS, IF ANY | |
|---|---|---|---|
| Embolus of iliac artery | 2 | | |
| (a) Emboli in both iliac arteries | | Bilateral embolectomy | 1 |
| (b) Embolus in right common iliac | | Embolectomy of right common iliac artery | 1 |
| Thrombus of femoral artery | 2 | Embolectomy | 1 |
| | | Lumbar sympathetic block | 1 |
| Pyelonephrosis and nephrolithiasis | 2 | | |
| Pulmonary neoplasm | 1 | Bronchoscopy | 1 |
| Mediastinal tumor, thymoma, or lymphoblastoma | 1 | Biopsy of axillary lymph node | 1 |
| Ruptured peptic ulcer or ruptured appendix | 1 | Right paramedian incision appendectomy | 1 |
| Pancreatic or peritoneal cyst | 1 | Exploratory laparotomy | 1 |
| Ruptured appendix | 1 | Exploratory laparotomy | 1 |
| Acute pancreatitis | 1 | Exploratory laparotomy | 1 |
| Abdominal mass | 1 | | |
| Renal colic | 1 | | |
| Subdiaphragmatic abscess or ruptured viscus | 1 | | |
| Embolus of right brachial artery | 1 | | |
| Partial intestinal obstruction | 1 | | |
| Gastrointestinal malignancy | 1 | | |
| Renal tumor | 2 | Exploratory laparotomy | 1 |
| Ruptured bowel | 1 | | |
| Total | 21 | | 11 |
| Total number of cases studied | | 278 | |
| Total number of cases diagnosed as surgical disease | | 21 (7.6%) | |

TABLE II. SURGICAL DISEASES CONSIDERED IN THE DIFFERENTIAL DIAGNOSIS

| DISEASES | NUMBER |
|---|---|
| Acute abdomen: Acute pancreatitis, cholecystitis, or perforated ulcer | 6 |
| Perforated peptic ulcer | 3 |
| Nephrolithiasis or kidney tumor | 4 |
| Subdiaphragmatic abscess | 1 |
| Mesenteric thrombosis | 1 |
| Total | 15 |
| Total number of cases in which surgical disease was considered in the differential diagnosis | 15 (5.4%) |

CASE REPORTS

## *Cases of Dissecting Aortic Aneurysms Diagnosed as Surgical Disease*

CASE 1.—McGeachy and Paullin,[9] 1937.

*Leading symptoms.*—A 39-year-old Negro, while stepping into his ice wagon, suddenly seized with a severe angonizing pain beneath sternum and radiating to right lower abdominal quadrant, also to back and right arm. Fell to street and because of weakness of right arm and leg, could not walk. Was carried to hospital. Admitted to surgical service with diagnosis of renal colic.

*Physical examination:* Well-developed, well-nourished man with temperature of 97° F.; pulse, 60; respiration, 28; blood pressure, 160/80. Heart: Enlarged to left, no murmurs. Systolic thrill and bruit over left inguinal region and left femoral artery pulsating vigorously No pulsations in any of vessels of right leg. Abdomen: Flat, with board-like rigidity; marked generalized tenderness especially in right lower quadrant and right inguinal region where soft irregular tender mass was felt, apparently beneath the skin. Deep reflexes absent in right leg. Skin of the right leg cold and clammy. Later diagnosis of dissecting aneurysm with compression of right iliac artery made. Had fairly comfortable night and died suddenly next day. White blood cells, 14,140; red blood cells, 2,090,000. Spinal fluid under slightly increased pressure. Chest x-ray showed cardiac enlargement and enlargement of thoracic aorta (no definite aneurysm).

*Differential Diagnosis.*—Renal colic (made on admission to surgical service); dissecting aneurysm.

*Ante-mortem Diagnosis.*—Dissecting aneurysm.

*Treatment.*—Symptomatic.

*Extent of Dissection.*—A 3 cm. transverse tear 1 cm. above valve cusps. Dissection included left carotid and subclavian arteries and down to and including right iliac artery. Blood had escaped behind pleural spaces and extended retroperitoneally into right inguinal region.

*Comment.*—Admitted to Surgical Service with diagnosis of renal colic. Diagnosed as dissecting aneurysm prior to death.

CASE 2.—Glendy, Castleman, and White,[12] 1937.

*Leading symptoms.*—A 52-year-old white male, laborer, past history incomplete. Known to have had generalized abdominal pain with nausea and vomiting for three days prior to entry. Subsequently, had precordial pain. Because of tender distended abdomen, was sent to hospital for surgical consideration. No record of blood pressure before entry.

*Physical examination:* Found to be acutely ill with pulse, 140; temperature, 101.6° F.; respiration, 34. Heart and lungs said to be normal. Blood pressure, 108/60. Abdomen: Very stiff and generally tender, but not more on one side than other. Reflexes normal. Before exploratory laparotomy could be done, he went into shock, blood pressure falling to 40 systolic. Sank rapidly into coma and died in spite of blood transfusion and other intravenous therapy. Terminal white blood cells, 34,000. X-ray film of abdomen gave no significant information.

*Differential Diagnosis.*—Generalized peritonitis, probably due to ruptured appendix.

*Ante-mortem Diagnosis.*—Generalized peritonitis, probably due to ruptured appendix.

*Extent of Dissection.*—A 4 cm. transverse tear in intima 1.5 cm. above aortic ring with dissection of entire aorta down to iliacs. Proximal dissection into myocardium around right coronary orifice. No external rupture.

*Comment.*—Died before exploratory laparotomy for generalized peritonitis, believed probably due to ruptured appendix, could be done. From history and physical report, diagnosis of dissecting aneurysm could not have been suspected.

CASE 3.—Glendy, Castleman, and White,[12] 1937.

*Leading Symptoms.*—A 44-year-old white man, salesman, while walking suddenly seized with severe pain over posterior aspect of right shoulder, which spread to left shoulder, down

anterolateral aspects of chest on both sides, down sides of abdomen, and finally settled and remained with greatest intensity in both lower abdominal quadrants. Legs became numb and cold, skin over them blue, and slightest movement caused great pain in right leg.

*Physical examination:* Patient obese, unable to walk, and in moderate shock. Marked cardiac enlargement with blowing systolic apical murmur. Rate regular at 104; blood pressure, 190/110. Lungs: Clear. Abdomen: Tympanitic. Both legs cold, later left leg warm again. Absent arterial pulsations and absent sensation, right leg. Embolectomy, right common iliac artery, 16 hours after onset. Clot 3 inches in length was removed but blood failed to flow from proximal segment. Condition poor and blood pressure 100/50, postoperatively. Developed pulmonary edema and died 36 hours after onset. Total hematuria in catheter specimen of urine taken on arrival at hospital. No other laboratory studies reported.

*Differential Diagnosis.*—Embolism of right common iliac artery (Surgical Service); dissecting aortic aneurysm (cardiac consultant).

*Ante-mortem diagnosis.*—Embolism of right common iliac artery (Surgical Service); dissecting aortic aneurysm (cardiac consultant).

*Treatment.*—Embolectomy, right common iliac artery.

*Extent of Dissection.*—A 5 cm. transverse tear at junction of arch and descending portion of aorta. Dissection down into left common iliac where it ruptured back through the intima into the lumen. An 8 mm. dissection into media of left renal artery.

*Comment.*—Died after embolectomy (right common iliac). Although patient operated upon for embolism by surgical service, diagnosis of dissecting aneurysm was made by cardiac consultant. Whether this diagnosis was made pre- or postoperatively is not stated.

CASE 4.—Glendy, Castleman, and White,[12] 1937.

*Leading Symptoms.*—A 51-year-old white man, teacher, seized with sudden, terrific, but dull pain over entire lower abdomen quickly spreading to both legs, chest, and shoulders. Within ten minutes was paralyzed from waist down. Numbness began in feet and rapidly ascended to point just above the symphysis pubis. Chest and shoulder pain rapidly disappeared but lower abdominal pain persisted. Pale, ashen cyanosis, with complete flaccid paralysis of both legs on physical examination. Feet and lower legs waxy, white, and cold. Skin above this to symphysis pubis bluish red and cool. Absent arterial pulsations bilaterally. Sensation and reflexes absent in lower extremities. Abdomen not tender nor distended. Blood pressure 220 systolic; pulse, 90 to 120; respirations, 20; systolic and diastolic murmurs all over precordium, loudest at base.

*Differential Diagnosis.*—Embolism, iliac arteries, bilateral.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—To operating room for proposed iliac embolectomy. Died during embolectomy.

*Extent of Dissection.*—T-shaped tear in intima 1.5 cm. above aortic cusp with dissection of media of whole aorta to 4 cm. above its bifurcation where massive clot within wall of vessel occluded lumen.

*Comment.*—Died on operating table during course of proposed embolectomy, iliac. Diagnosis could have been suspected because of nature of pain, presence of high blood pressure, neurological findings, and peripheral vascular changes.

CASE 5.—Hargrove,[13] 1939.

*Leading Symptoms.*—A 56-year-old Negro man, farmer, admitted April 30, 1935, complaining of generalized abdominal pain. Became ill at 2 P.M. on April 29, with sudden substernal pain radiating to back between shoulder blades, to abdomen and thighs. Immediately felt weak. Became dyspneic and had cold perspiration. Vomited shortly afterward. Pain continuous throughout night. On admission, had generalized agonizing abdominal pain with radiation to thighs.

*Physical examination:* Well-nourished man in great pain and very dyspneic. Comfortable in sitting posture. Pulse, 58; blood pressure, 220/100; respiration, 22; temperature, normal. Respirations labored and restrained. Chest: clear. Heart: enlarged to left and

systolic aortic murmur present. Abdomen: Very tender, reducible umbilical hernia, especially along upper right margin of its opening through abdominal wall. No abdominal distention or rigidity. X-ray view of gastrointestinal tract, negative. White blood cells, 8,000; Kahn, negative. Diagnosis of acute abdomen, ruptured appendix, or ulcer made. Appendectomy done under spinal anesthesia. On seventh postoperative day, complained of severe precordial pain, thought to be angina, and died suddenly on tenth postoperative day.

*Differential Diagnosis.*—Acute abdomen: ruptured appendix or peptic ulcer. Angina pectoris.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Appendectomy through right paramedian incision.

*Extent of Dissection.*—In arch of aorta at level of origin of left subclavian and common carotid arteries, there was a dissecting aneurysm which extended downward into anterolateral aspect of aorta, throughout length of aorta, and left common iliac artery. There was long slightly irregular longitudinal slit in the intima extending into the media, commencing at origin of left subclavian artery and extending downward for 5 cm. There was transverse extension from this for 1.5 cm. Dissection occurred in the plane of media, creating false passage admitting two fingers. There was rupture of aneurysm at its beginning into left pleural cavity. Numerous atheromatous plaques scattered throughout aorta.

*Comment.*—Diagnosis could have been suspected because of nature of pain associated with faintness and vomiting, presence of high blood pressure, and enlarged heart.

CASE 6.—Holland and Bayley,[10] 1940.

*Leading Symptoms.*—A 46-year-old Negro man admitted March 3, 1939. Several days previously was seized with sudden pain in lower abdomen radiating into right leg. At same time, noted mass increasing rapidly in region of right femoral canal. Abdominal pain subsided before admission. Pain in leg persisted and motion was impaired.

*Physical examination:* There was large, tender, firm, pulsating mass, just inferior to the medial third of the right inguinal ligament. Right thigh and leg cold and clammy. No arterial pulsations palpable. Urine, negative; Wassermann, negative; white blood cells, 12,500. Diagnosis of thrombosis of right femoral artery made and lumbar sympathetic block done. Patient died within two hours.

*Differential Diagnosis.*—Thrombosis of right femoral artery.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Lumbar sympathetic block.

*Extent of Dissection.*—Primary transverse tear of aortic intima located 2.5 cm. from aortic valve and encircled entire aortic lumen. Dissection extended proximally and distally over entire length of aorta. Secondary rupture into the right thigh, 7 cm. below inguinal ligament and into pericardial cavity.

*Comment.*—History and physical insufficient for diagnosis. However, when there is sudden onset of signs of occlusion of peripheral vessel and no reason to suspect embolus or thrombus as auricullar fibrillation, myocardial infarction or trauma, one should be suspicious of dissecting aneurysm.

CASE 7.—Stevenson,[14] 1938.

*Leading Symptoms.*—A 46-year-old farmer's wife, treated with opiates for past two years for pains resembling renal colic. Diagnosis of pyelitis and nephrolithiasis made by family doctor. In August, 1937, had pain in loin radiating into pelvis. Pain persisted for 10 days, becoming progressively more severe. On hospitalization, patient found to be thin woman, weighing 100 pounds, in acute distress. Temperature, 101° F.; pulse, 120. Large soft solid mass filled whole left groin, extending from costal margin to below crest of the ileum with lower rounded border overhanging pelvis, and extended also to midline of the abdomen. Red blood cells, 1,300,000; hemoglobin, 28 per cent; white blood cells, 36,200, with polymorphonuclears predominating. Stimulants and intravenous therapy of no avail. Patient died several hours after admission.

*Differential Diagnosis.*—Large pyelonephrosis with nephrolithiasis.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Stimulants and intravenous therapy.

*Extent of Dissection.*—Clot extended from arch of aorta down to pelvis. Small slit at junction of right aortic fifth intercostal artery was present. Opening was one-fourth inch long and extended through the intima and media. Thoracic aorta contained numerous atheromatous plaques and its diameter larger than normal. Surrounded by laminated clot, 1 to 2 cm. in thickness. Clot lay between media and adventitia.

*Comment.*—Cause of death, hemorrhage through dissecting aneurysm of the thoracic aorta. Opening of aneurysm was so small as to allow only repeated small hemorrhage. Condition existed for months, as evidenced by laminated clot. Only small amounts of fresh blood were able to trickle along path between the coats of aorta and through aortic opening into left retroperitoneal subdiaphragmatic space. From history and physical given, diagnosis of dissecting aneurysm could not have been made.

CASE 8.—Roach,[15] 1939.

*Leading Symptoms.*—A 52-year-old white woman with a past history of diabetes mellitus and peptic ulcer; took insulin. Gastrointestinal studies in 1927 showed hesitation of barium passage at lower end of esophagus as if there were constriction. Patient all her life was troubled with abdominal pains and distress. In 1928, and 1931, had episodes of hemoptysis. Admitted in May, 1937, complaining of severe paroxysms of pain in abdomen and back for past year. Pain in back was felt at different levels but radiated up as high as the angles of the scapulae and down well into the lumbar region. The attacks were severe and occurred at irregular intervals, frequently when eating. They lasted variable times, up to two hours. Lately had vomited with the pains.

*Physical examination:* Fairly well-nourished woman with moderate sclerosis of retinal and peripheral arteries. Lungs and heart normal. Blood pressure 170/80. Abdomen: Marked tenderness of epigastrium and upper part of abdomen; marked muscle spasm in this area. Pulsations in legs equal. X-rays of dorsal and lumbar spine were negative. Wassermann, negative. X-ray of stomach revealed medium residue in the cardiac end of stomach. Barium entered stomach freely. Extraneous to stomach along lesser curvature was palpable mass which elicited much pain with pressure. Duodenal bulb deformed due to mass. X-ray revealed nonfunctioning gall bladder. Patient continued about same with irregular periods of pain, and on January 20 she vomited a large amount of blood and died within few minutes.

*Differential Diagnosis.*—Abdominal mass.

*Ante-mortem Diagnosis.*—Not mentioned.

*Treatment.*—Symptomatic.

*Extent of Dissection.*—Not mentioned.

*Comment.*—The stomach, duodenum, and jejunum were filled with blood within 15 inches of ileocecal valve. No ulceration or free bleeding point found. Large clot in dissecting aortic aneurysm in contact with third portion of duodenum. This had eroded through duodenum and massive hemorrhage had occurred. Aorta showed marked reaction in walls; arteriosclerosis and organizing clot. Correct diagnosis probably not possible from history and physical given.

CASE 9.—Baker,[16] 1940.

*Leading Symptoms.*—On Dec. 22, 1936, after retiring, patient was awakened with severe dyspnea and dull aching pain across upper chest. Remained dyspneic for several days. Dyspnea and dullness gradually cleared up. X-ray examination showed pleural effusion at left base and slight bulging of right mediastinal shadow. Blood pressure was normal. There was always a tendency to cough, some dyspnea, and easy fatigability until March, 1939. At that time, while working in the field, patient suddenly stricken with dull aching pain across upper chest as though "heavy weight" pressed down on him. He became extremely weak and legs felt paralyzed; no definite dyspnea and no radiation of pain. The onset was extremely

sudden and he was forced immediately to the ground. He crawled home. At that time he was very pale and weak. Later in evening, he became cyanotic and dyspneic and was taken to hospital.

*Physical examination:* Acutely ill man, deeply cyanotic from the costal margins upward. Cervical veins and veins over anterior chest and abdomen markedly distended. Moderate hyperpnea, heart sounds not remarkable. Marked widening of mediastinal dullness; scattered râles over both lung bases. Blood pressure: 120/80 in arms and both radial pulses equal. Direction of blood in superficial vessels was reversed. At later examination, second aortic sound somewhat accentuated and soft blowing systolic murmur heard over this area; no diastolic murmur. Patient became more cyanotic and had severe coughing paroxysms associated with marked dyspnea. Complained of a constant pressure sensation in upper chest. Compensatory circulation became more evident. Bilateral hydrothorax developed. Pulses in lower extremities remained normal. Upper extremities edematous. Patient died April 17, 1939, during severe coughing paroxysm. On March 21, 1939, mediastinal shadow was considered to be propably a tumor of vascular origin. Electrocardiogram showed negative T-1 and negative T-2. White blood cells, 14,200 with normal differential. Biopsy of small axillary lymph node normal. Blood Wassermann and Kahn, negative.

*Differential Diagnosis.*—Nonmalignant neoplasm, probably thymoma and aortic aneurysm.

*Ante-mortem Diagnosis.*—Nonmalignant neoplasm; lymphoblastoma.

*Treatment.*—None mentioned.

*Extent of Dissection.*—Situated 3 to 5 cm. above sinus of Valsalva was a transverse laceration of the aorta measuring 6 cm. On right aspect of this laceration was longitudinal tear extending upward for 5 cm. This communicated with aneurysmal sac the size of a fist, which had descended downward between auricles and to some extent between aortic valve and pulmonary valve. It had encroached upon superior vena cava which was filled with antemortem thrombus.

*Comment.*—Anatomic diagnosis: dissecting aneurysm, ascending; thrombosis of superior vena cava; edema of shoulders, arms, and neck; bilateral hydrothorax. Sudden onset of severe chest pain with collapse and vascular and x-ray changes were suggestive of correct diagnosis.

CASE 10.—Reich,[17] 1944.

*Leading Symptoms.*—A 61-year-old white man with pain in chest radiating to back, lasted for two days.

*Differential Diagnosis.*—Gastrointestinal malignancy or coronary occlusion.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Not mentioned.

*Extent of Dissection.*—Tear just above aortic valve 3 cm. in length.

CASE 11.—Reich,[17] 1944.

*Leading Symptoms.*—A 54-year-old white man with pain in L.U.Q. radiating upward for seventeen days. Mass felt in left flank.

*Differential Diagnosis.*—Carcinoma of left kidney.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Not mentioned.

*Extent of Dissection.*—Retroperitoneal rupture at level of left kidney.

CASE 12.—Reich,[17] 1944.

*Leading Symptoms.*—A 72-year-old white man, pain over seventh thoracic vertebra radiating to shoulder and abdomen for 8 hours.

*Differential Diagnosis.*—Ruptured bowel; dissecting aneurysm.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Not mentioned.

*Extent of Dissection.*—Left hemothorax with tear from arch to below kidneys.

*Comment.*—Scar of healed ulcer in anterior wall of stomach.

CASE 13.—Reisinger,[18] 1940.

*Leading Symptoms.*—A 50-year-old white man with a past history of acute nephritis with uremia 29 years ago. No record of high blood pressure. For eight years he had peptic ulcer. Six months before admission noted shortness of breath with slight swelling of ankles. Five days before admission, patient worked harder than usual. Next day, had knifelike pains in chest and dyspnea at rest. Pain improved for few days with sedation, but symptoms recurred with pain, nausea, emesis, extreme dyspnea, and palpitation. On admission, had Cheyne-Stokes respiration and was fibrillating; blood pressure, 160/40. Next morning, pain in chest relieved, heart regular, blood pressure, 148/116. Heart sounds distant. White blood cells, 17,250; red blood cells, 3.5; hemoglobin, 11.2 Gm.; nonprotein nitrogen, 46.1 mg. Urine, albumin in several specimens and few white blood cells and red blood cells. X-ray of chest: Areas of infiltration or consolidation in lung fields; aorta unusually wide to right with prominent aortic knob; irregular dense shadow extended below the pulmonary artery. Heart: Not enlarged. Electrocardiogram showed evidence of myocardial damage but not characteristic of coronary occlusion. Patient improved and went home after two months with weakness, precordial pain, and dyspnea. Nine months later, had x-ray at bedside which revealed large shadow occupying two-thirds of the left side of thorax, rounded opacity projecting from cardiac and aortic shadow to right between second and fourth ribs anteriorly was noted. Diagnosis of tumor of lung made. Bronchoscopic examination was negative, but, nevertheless, x-ray therapy was given, and mass in left upper chest decreased in size. Right shadow remained unchanged. Patient continued to improve except for weakness and nausea at times. One week before death, complained of difficulty swallowing liquids, edema of ankles, and pain in chest. Had profuse hemorrhage from mouth and died.

*Lab. work terminally:* Red blood cells, 1.9; hemoglobin, 7 Gm.; urine, albumin, and red blood cells in all urine; nonprotein nitrogen, 78.9 mg.; creatine, 2.2 mg.

Media was split from beginning of aorta to arch with fissure extending up into great vessels of neck. On posterior portion of arch and along thoracic aorta, two sacs were within walls. One, extending to right in close apposition to esophagus and filled with laminated clot; other, projecting to left and containing fresher blood. No intimal tear present. Esophagus constricted by aneurysmal mass, and numerous punched-out ulcers were present in esophageal mucosa which might have communicated with aneurysm.

*Differential Diagnosis.*—Pulmonary neoplasm; dissecting aneurysm.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Not mentioned.

*Comment.*—Correct diagnosis was only slightly suspected for patient was bronchoscoped and given x-ray therapy to "tumor."

CASE 14.—Held,[19] 1944.

*Leading Symptoms.*—Nonsyphilitic 57-year-old man had following symptoms for several weeks: Pain in left hypochondrium radiating to left side of chest, moderate elevation of temperature and increased breath sounds over base of left lung with diminished respiratory mobility of left chest. X-ray of chest, negative. Persistent pain in left lower chest and subdiaphragmatic region and fluoroscopic findings of high dome of left diaphragm with restricted mobility of left diaphragm led observers to insert needle into left subdiaphragmatic space. No fluid obtained. Several weeks later, patient suddenly went into shock. Ruptured viscus was suspected. Blood transfusion and other restorative measures failed and patient died within few hours.

*Differential Diagnosis.*—Subdiaphragmatic abscess, ruptured viscus.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Transfusions.

*Extent of Dissection.*—Not mentioned. Autopsy revealed ruptured abdominal aorta with diffuse bleeding into peritoneal cavity. Pathologist demonstrated slowly dissecting aneurysm had preceded rupture.

*Comment.*—Diagnosis not possible from history and physical given.

CASE 15.—Leitch,[11] 1944.

*Leading Symptoms.*—A 63-year-old white man, while at work, tried to lift heavy piece of machinery and developed sudden pain in back and weakness of right arm lasting one-half hour. A few minutes later, he collapsed and was taken to hospital.

*Physical examination:* Patient in shock. Skin cold and clammy and mucous membranes pale. Respirations were rapid. Patient was fully conscious. Blood pressure: 190/90. Complained of weakness of right arm which was cooler than left, cyanotic, and had no pulsations. Heart sounds, distant but regular. Systolic and diastolic blow over precordium. Right carotid pulse palpable but no pulsations in axillary artery. Patient died one-half hour after admission.

*Differential Diagnosis.*—Embolism of right brachial artery, pulmonary embolism.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Morphia.

*Extent of Dissection.*—Dissecting aneurysm extended from base of heart down to and including 2 cm. of each common iliac artery. There was horizontal tear of intima, 2 cm. above aortic valve, and longitudinal tear of adventitia just above base of heart, with hemorrhage into pericardium.

*Comment.*—Diagnosis could have been suspected because of sudden onset of pain with collapse, peripheral vascular changes, and high blood presure.

CASE 16.—Clinicopathological Cases (Case 27292),[20] 1941.

*Leading Symptoms.*—A 57-year-old housewife with hypertension of 4 years' duration, systolic 230. Four hours before admission, smothering sensation developed in substernum without dyspnea, pain, or cough. One-half hour later, had gradually increasing pain and tingling in left leg followed by numbness, coldness, and paralysis of this extremity. Only transient tingling in right foot.

*Physical examination:* Patient pale, apprehensive, anxious, and acutely ill. Heart enlarged to left with systolic and diastolic murmurs heard best at base over aortic area. Regular rhythm. Blood pressure: 160/110. Occasional moist râles heard throughout chest. Abdomen: Negative. Patient unable to move left leg which was cold and mottled pink below line drawn horizontally to lower margin of vulva. No pulsations of femoral, popliteal, posterior tibial, or dorsalis pedis arteries were felt. Pulsations present throughout right leg. Two and one-half hours after admission, patient could move left ankle but not toes. Leg was warmer and pulsations palpable in femoral artery just below profunda femoris branch. Three hours after admission, incision was made along left femoral artery below Poupart's ligament under local. Patient died 9 hours after admission.

*Differential Diagnosis.*—Embolus of left femoral artery; hypertensive heart disease.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Embolectomy, left femoral artery.

*Extent of Dissection.*—In ascending aorta, several cm. above valve, was T-shaped intimal tear which communicated with dissecting aneurysm. External perforation occurred just below this region into pericardium which was full of fresh blood. Dissection continued down aorta and to left external iliac and femoral arteries to point 5 cm. below Poupart's ligament, at which point lumen was occluded by opposing intimal surfaces.

*Comment.*—Diagnosis might have been suspected because of hypertension, nature of pain, and peripheral vascular changes.

CASE 17.—Bauersfield,[2] 1947.

*Leading Symptoms.*—A 70-year-old white woman with no history of exertion prior to onset of slight epigastric pain with nausea and vomiting for three weeks. No dyspnea, no history of hypertension. Blood pressure: 118/75; no murmurs. Developed right hemiplegia with hyperactive reflexes which disappeared on second day. No chest x-ray or serology done. Flat plate of abdomen showed dilated loops of bowel representing partial intestinal obstruction. Blood cell count, normal.

*Differential Diagnosis.*—Partial intestinal obstruction.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Not mentioned.

*Extent of Dissection.*—Tear in the mesenteric artery 2 cm. from its origin, extended up superior mesenteric artery for 5.5 cm. Moderate degree of arteriosclerosis. Thrombus found occluding artery and produced gangrene in bowel.

*Comment.*—Dissecting aneurysm of superior mesenteric artery. Patient died of gangrene of bowel with intestinal obstruction.

CASE 18.—Reich,[17] 1944.

*Leading Symptoms.*—A 44-year-old white man, complaining of severe pressing precordial pain, followed in one-half hour by radiation into left side of abdomen and into arms and legs. Pain was cramplike and associated with nausea and dyspnea. Has had episodes of cramplike pains in extremities for about three months.

*Physical examination:* Tracheal tug and marked pulsations suprasternally. Heart enlarged to left, regular rhythm, no thrills or murmurs. $A_2$ accentuated. Blood pressure: 210/135. Abdomen: Spasm of abdominal muscles.

*Lab. examination:* Blood urea, 53; creatinine, 1.28; blood sugar, calcium, and Wassermann, normal. Agglutination tests, negative. Blood cell count, normal. Three weeks after admission, patient complained of sudden discomfort in left chest and examination revealed massive pleural effusion on left with displacement of heart and mediastinum to right. Patient gradually weakened and died six weeks after admission.

*Differential Diagnosis.*—(1) Tuberculosis, (2) malignancy of lung, (3) dissecting aneurysm, (4) nephrolithiasis due to hyperparathyroidism.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—No operation.

*Extent of Dissection.*—Irregular tear ¾ inch in length at junction of terminal portion of aortic arch and descending aorta. Adventitia was dissected off to point 1 inch above crura of diaphragm at which point another irregular tear of intima measuring ¼ inch was noted. Intimal surface was smooth and elastic with only few yellowish plaques scattered throughout. Superior tear dissected media into large space overlying mesial portion of left upper lobe. This space was filled with clotted blood.

*Comment.*—Left hemothorax.

CASE 19.—DeAngelis,[21] 1943.

*Leading Symptoms.*—A 64-year-old white man, with severe recurring, radiating pain of three years' duration. Pain described as "agonizing and viselike," and began in epigastrium or lower substernal region and radiated over entire chest and back and down arms as far as finger tips, also, upward into head. Radiation fairly constant. At first these occurred at irregular intervals after strenuous exertion and lasted only few minutes. Later, they became more frequent and severe, lasting thirty minutes or longer. For eighteen months a mass in abdomen was evident and increasing in size.

*Past history:* History of chronic atrophic gastritis with achylia gastrica, bilateral direct inguinal hernia, hypertension, and severe arteriosclerosis.

*Present illness:* Tall, thin, elderly chronically ill white man. Lungs: clear. Heart: enlarged to the left, normal rhythm, systolic and diastolic murmurs in aortic region, transmitted along right sternal margin. Pulse, 76; blood pressure, 250/170; left arm; 256/174, left leg. Abdomen: Tumor size of grapefruit just below epigastric region, tense, immovable, not pulsating. No thrill, but inconstant systolic murmur audible. Direct inguinal hernia present on each side. Arteries, hard and tortuous. Blood cell count, normal. Wassermann, negative. Urine, occasional hyaline cast. X-rays: Gastrointestinal series, external pressure over lesser curvature of stomach; chest, enlargement of cardiac shadow especially left ventricular portion, and greatly dilated and tortuous aorta.

During first five days in hospital, patient was up and about complaining of constant dull abdominal pain. Diagnosis of pancreatic or peritoneal cyst or possible abdominal aneurysm

of aorta was made. Last diagnosis apparently had little favor, for patient was operated upon and dissecting aneurysm found. No surgical procedure done. Patient died on third postoperative day after suddenly experiencing severe pain in abdomen.

*Differential Diagnosis.*—Pancreatic or peritoneal cyst, abdominal aortic aneurysm.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Exploratory laparotomy.

*Extent of Dissection.*—Abdominal cavity contained great quantity of coagulated blood. Abdominal aorta presented a large rent at its beginning, extended downward several centimeters beyond level of renal vessels. Entire vessel had been diffusely enlarged and anterolaterally the wall showed sheath dissection from region above renal vessels to just above bifurcation. Adventitial portion of aneurysm was over 10 cm. in diameter and contained large laminated thrombus. At its proximal level, it communicated with lumen of aorta through large opening. Internal surface of aorta showed marked atheromatous degeneration and ulceration throughout, especially in abdominal portion. Small ulcers were also present. Thoracic aorta was markedly dilated and tortuous.

*Comment.*—Correct diagnosis might have been suspected because of type of pain, existence of hypertension, and x-ray findings of greatly dilated and tortuous aorta.

CASE 20.—Henley and Gariepy,[22] 1946.

*Leading Symptoms.*—A 37-year-old white man, after a night of drinking, was awakened by severe chest pain about level of manubrium which gradually migrated to epigastrium. Relieved by morphine. Later in day was up and about. That night complained of generalized abdominal pain and discomfort in costal regions. Could not void, and was catheterized, which afforded some relief. Early in morning of Jan. 28, 1946, abdominal pain became very severe, particularly on left side about kidney region. No knowledge of hypertension. Negative serology.

*Physical examination:* Exsanguinated man. Blood pressure: 40/?; pulse, imperceptible; heart tones faint but regular; heart not enlarged. Chest, clear. Abdomen slightly distended; marked tenderness without rigidity in all quadrants.

*Lab. data:* Red blood cells, 2.6; hemoglobin, 55 per cent; white blood cells, 27,350; urine, negative. Electrocardiogram, sinus tachycardia; left axis deviation. Blood amylase and lipase, normal. Clinical diagnosis of acute pancreatitis was suspected. Given blood with no improvement; taken to operating room on Jan. 28, 1946, and under local anesthesia, large retroperitoneal hematoma was found. Patient developed severe shock and died 10 hours later.

*Differential Diagnosis.*—Acute pancreatitis.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Exploratory laparotomy.

*Extent of Dissection.*—There was a 1.5 cm. transverse tear in posterior wall of aorta 2 cm. above aortic orifice. Apparently perforation extended into pericardial sac resulting in hemopericardium. Grayish-yellow plaques were found over entire intima of ascending aorta. There was complete dissection of entire thoracic aorta from site of intimal tear just mentioned. This resulted in production of one tube within another, which were separated from each other by small amount of blood. Dissection extended into great vessels of aortic arch. Abdominal aorta was likewise dissected. Dissection had extended into mesenteric, renal, and iliac arteries.

*Comment.*—Anatomic diagnosis: Dissecting aneurysm of aorta extending into greater branches, atheromatous and medial degeneration of aorta, hemopericardium, cardiac hypertrophy and parenchymatous degeneration, moderate coronary sclerosis, bilateral serohemorrhagic hydrothorax, "nutmeg" liver and slight nephrosclerosis. Sudden severe pain in chest migrating to epigastrium might have made one suspicious of correct diagnosis, but other findings were not diagnostic.

CASE 21.—Carlson,[33] 1945.

*Leading Symptoms.*—A 27-year-old soldier admitted three weeks after wound by enemy sniper bullet. Bullet entered site between third and fourth lumbar spinous processes and

just slightly to right; wound healed. Patient had definite evidence of lesion of cauda equina involving mainly the lumbar group from 12 down to and including all sacral nerves. Day after admission he had sudden severe pain in right kidney region. Pain radiated anteriorly into right flank and groin. No radiation into genitals. Pain aggravated by lying on right side and relieved by lying on back or left side.

*Physical examination:* There was right costovertebral tenderness and tenderness in right side of abdomen. X-ray was negative for renal calculus. Considerable gas formation was seen throughout entire intestines and particularly over right kidney region. Few days after occurrence of pain, patient passed some blood in urine. On tenth day, showed signs of hemorrhage with secondary anemia. Abdomen explored. Large pulsating retroperitoneal mass found. Not possible to ligate aorta below renal arteries. Diagnosis of dissecting aneurysm of abdominal aorta was made. Patient died 11 days after operation.

*Differential Diagnosis.*—Dissecting aneurysm of aortic arch; renal calculus or tumor.

*Ante-mortem Diagnosis.*—Dissecting aortic aneurysm.

*Treatment.*—Morphine sulfate, opium, and rest.

*Extent of Dissection.*—Not mentioned.

*Comment.*—Diagnosis was not made preoperatively, but was made ante-mortem. There was no pathologic report.

*Cases of Dissecting Aortic Aneurysms in Which Surgical Disease Was Considered in the Differential Diagnosis*

CASE 1.—Osgood, Gourley, and Baker,[23] 1936.

*Leading Symptoms.*—A 50-year-old white man, following a heavy meal, was seized with steady, sharp epigastric and lower chest pain radiating to right and left sides of abdomen, to back of chest and into left flank. Pain was diffuse and poorly localized. No vomiting at this time but nausea. Pain subsided but soon recurred with greater intensity and remained. Noted dizziness. Vomited 5 hours after onset, and 6 times in next 4 hours.

*Physical examination:* Blood pressure: 182/88; equal bilaterally. Well-developed, well-nourished man in obvious pain turning from side to side. Very apprehensive. Face flushed, not cyanotic, no dyspnea, pulse 98 and regular, temperature 100° F. Systolic pulsation visible in carotid vessels and palpable in suprasternal notch and over left subclavian artery. Heart enlarged to left, increased upper sternal dullness, heart tones clear and sharp with long blowing systolic murmur over mitral area. Moderate resistance over upper abdomen, particularly on left side, but no localized tenderness. Peripheral vessels showed equal normal pulsations.

*Laboratory:* Icteric index, 7; urine, 2 plus albumin, 4 plus acetone, and diacetic acid. White blood cells, 9,900. Negative electrocardiogram. Treated with sedatives and analgesics. Discharged 59th day. Two months later, returned with orthopnea, flushed face, and complaining of severe chest pain. Blood pressure: 135/110; trachea deviated to right. Died 5 days after admission.

*Differential Diagnosis.*—Dissecting aneurysm of aorta; coronary thrombosis; and acute abdominal disease.

*Ante-mortem Diagnosis.*—Dissecting aneurysm.

*Treatment.*—Symptomatic.

*Extent of Dissection.*—Pericardial tamponade due to hemorrhage from intrapericardial performation of recent dissecting aneurysm confined to ascending aorta. Also, old dissecting aneurysm extending from arch of aorta into common iliac arteries.

CASE 2.—Glendy, Castleman, and White,[12] 1937.

*Leading Symptoms.*—A 53-year-old white man, laborer, while bending over fixing a pipe, suddenly seized with severe stabbing pain in left chest just below and outside of nipple. Pain increased in intensity, radiating to back and right chest. Worse on lying down and associated with dyspnea.

*Physical examination:* Anxious facies, great distress, few râles at left base of lung. Heart: Apical sounds faint, no audible sounds at base, regular rhythm, pulse, 120, systolic murmur over apex. Blood pressure: 190/120. Extreme epigastric tenderness. Respiration, 35.

*Course:* Agonizing seizures continued. Patient became hoarse and later lost voice entirely. Blood pressure fell to 100/78 after venous section. Intractable distention developed and surgical abdomen considered. Although in extremis, blood pressure remained elevated (160) and pulse very strong until one hour before death on sixth day.

*Laboratory:* White blood cells, 18,500; urine, trace of albumin; nonprotein nitrogen 67 mg. per cent.

*Differential Diagnosis.*—Pneumonia, chronic nephritis, arteriosclerosis, "surgical abdomen," and subdiaphragmatic abscess.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Not mentioned.

*Extent of Dissection.*—Elongated crevice in aortic intima at junction of aorta at ascending portion in region of arteriosclerotic ulcer with dissection of media of irregular contour extending down as far as inferior mesenteric artery. Rupture into mediastinum and both pleural cavities. Moderate arteriosclerosis of aorta.

*Comment.*—Nature of pain and elevated blood pressure were suggestive of dissecting aneurysm.

CASE 3.—Weiss,[24] 1938.

*Leading Symptoms.*—A 40-year-old man, Filipino, who had epigastric distress for seven years, six months before seen in December, 1936, had subcardiac and more severe epigastric distress, dyspnea, and palpitation on exertion. Discomfort appeared after eating, nonradiating and relieved by soda. Vomited coffee-ground material and had tarry stools.

*Physical examination:* Slight sclerosis of eye grounds. Heart slightly enlarged. Regular rhythm with soft systolic murmur. $A_2$ ringing. Arterial pressure, 168/106; 178/120. Urine negative; red blood cells, 2,400,000; hemoglobin, 34 per cent; white blood cells, 13,800. Stool tarry with 4 plus guaiac. Negative serology. X-ray of gastrointestinal tract: Gastric and duodenal ulcers. Electrocardiogram, digitalis effect. Treated with Sippy diet and iron, and patient improved. Diagnosis: Peptic ulcers, essential hypertension, and slight arteriosclerosis.

In November, 1937, at 1:30 A.M., experienced breathlessness and orthopnea for several hours. In afternoon of same day, developed sharp burning, boring, nonradiating pain in epigastrium. Entered hospital two days later, weak and irritable. Radial pulse imperceptible and arterial pressure not measurable. Heart moderately enlarged. Pulse, 130-140 and regular. Sounds weak. $P_2$ accentuated; $A_2$ barely audible. Blood pressure: 100/55. Lungs, normal. Tenderness over epigastrium but no spasm. Other findings irrelevant. Red blood cells, 6,000,000; hemoglobin, 95 per cent; white blood cells, 27,600 and 21,300. Nonprotein nitrogen, 46 mg. X-ray of chest: Widening of aorta. Electrocardiogram: Evidence of myocardial disease.

Condition remained unaltered for two days after admission. Patient suddenly collapsed, developed convulsions, and died.

*Differential Diagnosis.*—Coronary occlusion; perforated gastric ulcer.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—None mentioned.

*Extent of Dissection.*—About 1 cm. above aortic valve was sharp cleftlike rupture approximately 4 cm. in length, involving intima and media. Near by was another slit in aorta about 1 cm. in length. From base of aorta wall was dissected down to mouth of celiac artery, where dissection reruptured into aorta. Below this point aorta was intact. Small extravasation of blood occurred into posterior mediastinum. Scattered over intima of entire aorta were small, raised yellowish atheromatous deposits.

*Comment.*—Anatomical diagnosis: dissecting aneurysm of aorta with hemopericardium, pulmonary congestion, and edema; compression of mouth of left coronary artery caused by dissection of this artery; myocardial infarct of left ventricle; healed chronic pyelonephritis; moderate atherosclerosis of aorta, and healed cholecystitis.

CASE 4.—Nichaus and Wright,[25] 1940.

*Leading Symptoms.*—A 49-year-old Negro porter seized with sudden severe pain while standing on platform waiting for passengers. He was prostrated and broke out in "cold sweat." Pain radiated to neck and jaw, then penetrated to back in lower thoracic region, then to lumbar region and back of thighs as far as knees. In one hour pain localized in epigastrium and lower sternal region, radiating to interscapular region and along both costal margins. Pain persisted at this site until death 21 hours after onset. Pulse, 60; blood pressure, 140/100. Marked tenderness with muscle spasticity of epigastrium and right upper quadrant, simulating upper abdominal disease.

*Physical examination:* Robust, obese, Negro, weighing 200 pounds, critically ill. Face suffused and veins of neck moderately distended. Slight respiratory difficulty. Lungs: Clear. Heart: Apex in anterior axillary line; regular rhythm. Diastolic murmurs over aortic area along sternal border. Urine: Albumin, 2 plus; numerous pus cells. Hemoglobin, 15.6 Gm.; red blood cells, 4.7; white blood cells, 9,000; icterus index, 6. Wassermann test, negative. Electrocardiogram: Evidence of coronary thrombosis.

*Past history:* 1932—blood pressure, 230/140; 1936—blood pressure, 200/130.

*Differential Diagnosis.*—Acute abdomen; coronary thrombosis.

*Ante-mortem Diagnosis.*—Coronary thrombosis.

*Extent of Dissection.*—Irregular triangular rupture 1.5 cm. in length and about 2.5 cm. above aortic valve on right lateral surface slightly anterior and extended through intima and about four-fifths of media. From rupture, blood burrowed toward heart involving most of circumference of aorta and then extended subepicardially over right auricular surface. The intramural hematoma extended distally to level of fourth thoracic vertebra involving one-fourth to one-half of circumference of aorta. Minimal sclerosis seen. Torn edge showed normal thickness of media and minimal intimal thickening. At junction of ascending and transverse arch of aorta were 4 to 5 peculiar longitudinal grooves, measuring 1.5 cm. in diameter. These reduced size of lumen considerably. Beyond this, arch widened. No atheromatous ulcers seen.

*Comment.*—Pericardial sac, 1,000 c.c. of clotted blood. Coronaries patent. Nature of pain and elevated diastolic blood pressure and brief duration of the illness are suggestive of dissecting aneurysm.

CASE 5.—Finkelstein and Jacobi,[26] 1940.

*Leading Symptoms.*—A 54-year-old white man complaining of constant pain in epigastric region of one week's duration. Pain was severe, unremitting and radiated directly to back. Not relieved by alkalies or food and became progressively worse until day of admission. Patient was nauseated. No hematemesis or melena. For past 8 years until one week ago, epigastric pain was relieved by food and soda.

*Physical examination:* Well nourished, head and upper chest flushed. Lungs: Dullness over both bases, few crepitant râles heard in left base in axillary region. Heart: Not enlarged; rate, 100; sounds fair quality; no murmurs. Blood pressure: 190/110. Abdomen: Marked tenderness in midepigastrium toward right of median line; tenderness also over 12th dorsal vertebra, posteriorly. Diagnosis of ulcer penetrating posteriorly into pancreas made.

*Laboratory:* Red blood cells, 5,200,000; white blood cells, 10,000; hemoglobin, 95 per cent; differential, normal; urine, negative; Wassermann test, negative. No occult blood.

*Course:* On second day of hospitalization, patient still had pain in epigastrium radiating to back, although he received tincture of belladonna and tincture of opium. On third hospital day, pain in epigastrium to back was so severe and tenderness so marked that perforation of ulcer posteriorly was feared. On sixth hospital day, epigastric pain radiated to back was still present. On seventh hospital day, more comfortable. On tenth hospital

day, x-ray of stomach and duodenum revealed no evidence of gastric or duodenal ulcer. Fluoroscoped again. Fusiform pulsating dilatation of entire descending aorta seen. Dissecting aneurysm suspected. On seventeenth hospital day, patient felt fairly well. At 7:15 P.M., while on bedpan, suddenly turned white, vomited, gasped for breath, and died.

*Differential Diagnosis.*—Perforating peptic ulcer, probably posteriorly into pancreas; dissecting aneurysm.

*Ante-mortem Diagnosis.*—Dissecting aneurysm.

*Treatment.*—Milk and cream, tincture of belladonna and opium, and morphine sulfate.

*Extent of Dissection.*—The ascending aorta showed little arteriosclerotic change. In arch of aorta, distal to openings of left subclavian and left carotid artery, was transverse linear break encircling posterior half of aorta and extending through intima into media. Aorta in its thoracic portion presented blood clot, laminated and partially organized, about 3 cm. thick, lying outside of media and pushing both intima and media into folds in lumen. Both thoracic and abdominal portions of aorta had lost much of their elasticity and contained many lipoid plaques, some of which were calcified. Area of atheromatous softening was present near break in arch of aorta.

*Comment.*—No evidence of recent or old ulceration of stomach or duodenum.

CASE 6.—Lowell, Jr.,[27] 1940.

*Leading Symptoms.*—A 50-year-old white man, known hypertensive, 36 hours prior to admission, while carrying a 40 pound weight in his arms, was suddenly seized with "knifelike" pain at tip of right scapula. It was severe enough to take his breath away, followed by feeling of weakness and faintness. Six hours following onset, pain appeared in midepigastrium extending down the costal margin on the left and into medial portion of left thigh with feeling of numbness in left leg. He was unable to lie on left side and unable to hyper-extend legs without severe pain in epigastrium. Vomited twice 8 hours prior to admission. Walked to hospital in "stooped" position.

*Physical examination:* Middle-aged man lying in "doubled-up" position on right side with moderate dull pain in epigastrium radiating into left iliac fossa and leg with feeling of numbness in leg. Left pupil was larger than right. Retinal examination showed marked arteriosclerotic changes. Chest, negative. Icteric tinge to skin and sclera. P.M.I. in anterior axillary line. No rub or murmur heard. Radial and apex rate same; radials bilaterally, regular and forceful at 80 per minute. Peripheral vessels markedly sclerotic. Femoral pulsations equal. Blood pressure: Right arm, 168/88; left arm, 180/98; right leg, 190/100; left leg, 196/106. Slight tenderness in midepigastrium and over aorta. Marked voluntary spasm in left upper and left lower quadrants. Neurological examination, negative.

*Laboratory data:* White blood cells, 21,550. Hemoglobin, 13.5 Gm. Stools, negative. Sputum, negative. Serology, negative. Urine: Fixed specific gravity with trace of albumin recorded consistently and few granular casts. X-ray view of abdomen, negative. Heart enlarged in its transcardiac dimensions, otherwise normal. Electrocardiographic tracing showed evidence of myocardial infarct. After failure to make diagnosis of acute abdomen, patient was accepted on medical service as case of dissecting aortic aneurysm. On ninth hospital day, he sat up in bed, called for an orderly, and was found cyanotic and gasping for breath. Died immediately.

*Differential Diagnosis.*—Acute surgical abdomen; dissecting aneurysm.

*Ante-mortem Diagnosis.*—Dissecting aneurysm.

*Treatment.*—Opiates.

*Extent of Dissection.*—Dissecting aneurysm in medial portion of aorta, extending from ascending aorta around arch and down descending aorta through abdominal portion into common iliac arteries. There were two ruptures of intima into media. Inferior one of these was presumably the first. Rupture was seen on anterior aspect of descending aorta, immediately below junction of left subclavian artery. Superior laceration was seen in ascending aorta at the beginning of arch. There was connection between medial cavity below inferior laceration and right pleural cavity. Latter was filled with blood.

*Comment.*—Anatomic diagnosis: Dissecting aneurysm of thoracic and abdominal aorta and right hemothorax. Although the admission diagnosis was incorrect, after complete checkup done, correct diagnosis made.

CASE 7.—Reisinger,[18] 1940.

*Leading Symptoms.*—A 41-year-old Negro, after an evening meal several days before admission, suddenly became nauseated and vomited. Had pain in upper region of abdomen which disappeared. Took castor oil and pain returned. Went to hospital where enema was given and after four hours' observation, allowed to go home. Nausea and abdominal discomfort continued and went to family physician, who gave medication for "ptomaine poisoning" with no relief. Had coughing spell which increased pain over upper portion of abdomen, especially on right side. Had nausea, vomiting, and felt as if he had "gas on the stomach." First examiner impressed by abdominal symptoms and signs. Abdomen was tender about umbilicus, and muscle of right side slightly rigid. Temperature, 99.2° F.; pulse, 60; white blood cells, 9.5. Few cells in urine. Blood pressure, 150/90. Heart not enlarged and regular rhythm. Peripheral arteries thickened. Following month in hospital, patient felt poor. Had inconstant pain in epigastrium extending around right lumbar region and down spine, from interscapular region to coccyx. Cold water increased pain and after eating, often became nauseated and regurgitated food. Was constipated and passed only small quantities of urine. Temperature ranged from 100 to 101° F. Red blood cells, 4,400,000; hemoglobin, 14 on admission. Gall bladder studies, negative. Agglutination tests, negative. X-ray of chest, heart enlarged with widening of supracardiac shadow. Definite projection of first part of aorta to right, arch wide, knob broad and rounded. Descending aorta denser than usual and tended to fill concavity above left ventricle. Aortic outline appeared ragged. Trachea displaced to right. Electrocardiogram, normal. Toward end of second month, nausea and belching returned and there was pain in left side of chest, abdomen, and back. Red blood cells, 3,100,000; hemoglobin, 9.8 Gm.; white blood cells, 17,900. Repeat x-ray of chest: Cardiac shadow very large and supracardiac shadow larger. Right border of aorta formed wide arch. Aortic knob was less prominent and left supraventricular concavity was obliterated. In third month, patient had sudden attack of pulmonary edema, dyspnea, and shock. Heart enlarged with gallop rhythm, transient systolic murmur, and accentuated second sound at base. Had several such attacks over 48 hours. Third x-ray, greater widening of aorta, especially in descending portion. No pulsations felt in popliteal, dorsalis pedis, and posterior tibial arteries, although femoral pulses palpable. Percussion note left of spine from fifth thoracic to ninth thoracic was flat and loud rough systolic murmur was heard in this region only. Had gradually increasing heart failure and died 93 days after first symptom.

*Differential Diagnosis.*—Surgical abdomen; gall bladder disease, especially intestinal tuberculosis; coronary occlusion; dissecting aneurysm, second month.

*Ante-mortem Diagnosis.*—Dissecting aneurysm of aorta.

*Treatment.*—Analgesics.

*Extent of Dissection.*—Descending aorta presented transverse tear of intima across entire circumference of vessel, about 10 cm. below arch. Blood accumulated within split coats of aorta throughout thoracic and abdominal length of aorta. About 5 cm. above bifurcation into iliac artery was second tear in intima where more saccular aneurysm, not connected with previous one and containing older, laminated clot, extended along iliac arteries to Poupart's ligament. No evidence of erosion. Wall of abdominal aorta blackish and soft.

*Comment.*—Pericardial sac contained 200 c.c. of bloody fluid. Hemorrhage apparently originated in adherent area at reflection of pericardium from aorta. Decline in hemoglobin significant perhaps; x-rays and peripheral vascular changes made diagnosis.

CASE 8.—Finkelstein and Jacobi,[26] 1940.

*Leading Symptoms.*—A 54-year-old white man, admitted Oct. 8, 1937, complaining of constant pain in epigastric region for one week. Pain was severe, unremitting, and radiated directly to back. Not relieved by alkalies, food, or small doses of morphine sulfate and became progressively worse up to admission.

*Past history:* For 8 years patient suffered from gastric disturbance, diagnosed as peptic ulcer. Until one week ago, each attack was relieved by food and soda.

*Physical examination:* Well nourished, head and upper chest flushed. Temperature, 101° F.; eye, ear, nose, and throat examination, negative. Lungs, dullness noted over both bases, a few crepitant râles heard in left base in axillary region. Heart not enlarged, rate 100 per minute; fair quality, no murmurs. Blood pressure: 190/110. Abdomen: Marked tenderness in midepigastrium toward right median line. Tenderness also over twelfth dorsal vertebra posteriorly. Diagnosed on admission as peptic ulcer penetrating posteriorly into pancreas; treated with food and soda.

*Laboratory data:* Red blood cells, 5,200,000; white blood cells, 10,000; hemoglobin, 95 per cent. Differential, normal. Urine, negative. Blood glucose, 107 mg. per cent. Urea nitrogen, 27.5 mg. per cent. Blood Wassermann, negative.

*Course:* On Oct. 10, 1937, still had pain in epigastrium radiating to back. Marked tenderness in epigastrium still present. On October 11, pain in epigastrium radiating to back became so severe and tender that perforation of ulcer considered and surgeon consulted. The latter advised sedation, roentgen study of stomach followed by operation. On October 14, patient had epigastric pain radiating to back, but milder. On October 17, much more comfortable and gastrointestinal series done on October 18, showing no ulcer. On October 21, refluoroscoped. Noted fusiform pulsating dilatation of entire descending aorta. Dissecting aneurysm suspected. On October 25, patient felt "fairly well." While on bedpan at 7:15 P.M., patient turned white, vomited, gasped for breath, and died. From third day to death, temperature fluctuated between 100 and 102° F.

*Differential Diagnosis.*—Dissecting aneurysm; perforated peptic ulcer.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Food, soda, tincture of belladonna and opium, morphine sulfate.

*Extent of Dissection.*—Distal to openings of left carotid and subclavian arteries, was transverse linear break encircling posterior half of aorta and extending through intima into media, permitting escape of blood from within lumen of aorta into wall out beyond media. Aorta in its thoracic portion presented blood clot, laminated and partially organized about 3 cm. thick, lying outside of media and pushing both media and intima into folds into lumen. At region of break in arch, blood was present in loose perivascular tissue resting against very thin parietal pleura of apical portion of left chest. In its pulmonary aspect this pleura was coated with blood and fibrin. Both thoracic and abdominal portions of aorta had lost much of their elasticity and contained many lipoid plaques, some of which were calcified. An area of atheromatous softening involving both intima and media was present near break in arch of aorta. Renal, gastric, and splenic arteries showed moderate arteriosclerosis.

*Comment.*—Left pleural cavity contained one liter of sanguineous serum and several huge clots. Conservatism on part of consulting surgeon, for lack of definite diagnosis and refluoroscopy prevented unnecessary operation and made correct ante-mortem diagnosis.

CASE 9.—Levitt, Levy, and Cole,[28] 1940.

*Leading Symptoms.*—A 51-year-old white man, fireman, 8 hours prior to admission was awakened with sharp, excruciating pain, diffusely throughout lower abdomen, but more pronounced about umbilicus. Pain radiated through to back and at onset was accompanied by emesis. No blood in emesis; stools normal.

*Past history:* Precordial pain for 3 years, brought on by exercise, occurring 1 or 2 times monthly, lasting one to two minutes.

*Physical examination:* Blood pressure: 220/110. On admission, well developed, well nourished, in acute distress. Temperature, 97.2° F., pulse, 88; respirations, 30. No cyanosis. Chest, expansion limited on right; resonant throughout; breath sounds suppressed in right base. Heart enlarged to left, regular, $A_2$ accentuated. Blood pressure: 220/124. Abdomen: Resistance to palpation in right upper quadrant. Tenderness noted throughout, more over gall bladder region. Extremities, negative. Placed on surgical ward for observation

because also had hemoptysis and pain in chest on deep inspiration. Urine, negative; white blood cells, 22,250 (92 per cent polys). Pain and hemoptysis persisted. Six hours later pain still present. Temperature, 101.2° F.; pulse, 136; blood pressure, 140/92 left; right, 170/100. Heart enlarged, regular rate, tones suppressed. "G"-string type of friction rub in fifth interspace to left sternum. Abdomen: Fullness and spasm with no definite tenderness in R.U.Q. Slight cyanosis of nails. Electrocardiogram: Left axis deviation and myocardial damage. X-ray: Enlarged heart with aorta normal. Death on Jan. 11, 1939.

*Differential Diagnosis.*—Hypertensive cardiovascular disease; cardiovascular syphilitic aneurysm; coronary thrombosis; surgical abdomen—perforated ulcer or acute pancreatitis; dissecting aneurysm.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Opium, rest, and observation.

*Extent of Dissection.*—Aorta split into two layers. This split extending to base of heart where pericardium was attached to aorta. Large amount of dark blood between these layers. This space communicated with large pericardial space filled with blood. Aorta showed numerous thickened areas of raised yellowish plaques. One communicated with split space.

*Comment.*—Left pleural cavity—1,400 c.c. thin yellowish-brown exudate, plus clots and fibrous tags. Abdominal viscera, normal. Anatomic diagnosis: Aortic arteriosclerosis with dissection between coats and rupture into pericardial pocket.

CASE 10.—Buckley,[29] 1940.

*Leading Symptoms.*—A 43-year-old man with painless hematuria of two weeks' duration. One month previously, patient in hospital with complaint of severe pain which started beneath upper sternum, passed downward through chest and abdomen, centering posteriorly in lumbar muscles along spine. Marked numbness of left leg with inability to move left foot. Blood pressure: 230/130, low grade fever. White blood cells, 12,000 with 80 per cent polys. Wassermann and Kahn, negative. Cardiographic report, diffuse increase in caliber and density of aorta. Neurological signs subsided after two weeks and patient was discharged. Cystoscopy: Old clots evacuated from bladder. Right kidney, normal; left kidney, bloody urine dripped. Right pyelogram normal; left, large filling defect in pelvis with obliteration of superior calyx with some involvement of middle calyx. Kidney tumor suspected. Five days later, left pyelogram repeated showing pelvis smaller than in previous examination with fair visualization of superior calyx. Sent home and treated for hypertension. After hematuria stopped (one month) he again entered hospital for left pyelogram which was normal, then sure that hemorrhage was due to bleeding into left kidney and not due to tumor. Continued to work with occasional episodes of hematuria. Twenty-seven months after onset of symptoms, patient experienced severe headache followed by coma and death in few hours.

*Differential Diagnosis.*—Hypertension; cerebral hemorrhage; kidney tumor, left.

*Ante-mortem Diagnosis.*—Cerebral hemorrhage.

*Treatment.*—Not mentioned.

*Extent of Dissection.*—Old, healed dissecting aneurysm which began at level of orifice of left subclavian artery. Below this point aorta showed double channel, aneurysmal channel passed downward having ruptured all of intercostal and lumbar arteries on left, and had penetrated left renal, superior mesenteric, and right iliac artery. There was small communication between two channels just anterior to orifice of left renal artery.

*Comment.*—No examination of central nervous system permitted. Hematuria due to extension of aneurysm into left renal artery.

CASE 11.—Buckley,[29] 1940.

*Leading Symptoms.*—A 61-year-old man, while stooping, suddenly seized with pain originating in left lumbar area and radiating forward toward epigastrium. Pain characteristic of renal colic, although urogram revealed no calculus. Urine showed occasional red blood cells. Radiation of pain began to shift to L.L.Q. and patient entered hospital.

*Physical examination:* Patient in pain, anxious, with moderate pallor. Blood pressure: 190/110. Heart enlarged with no murmurs or irregularities. Temperature, 100. White blood

cells, 10,800 with 80 per cent polymorphonuclears. Wassermann and Kahn, negative. Urine, few red blood cells and white blood cells. Slight tenderness over left kidney, otherwise abdomen was negative. Cystoscopy: Bloody urine with clots found. No ulcers or tumors. Ureters: Left, urine was blood tinged; right, negative. Right pyelogram, negative; left, filling defect in pelvis with distortion and blunting of calices. Impossible to determine whether dealing with tumor of renal pelvis or blood clots. Following day, patient experienced severe pain due to passage of blood clots down left ureter. Large size catheter placed into left pelvis for drainage. Although catheter draining well, patient complained of spasmodic, severe pain in left epigastrium, occasionally radiating to left lumbar area and left lower quadrant. There occurred periods of cyanosis, pallor, and anxiety, together with variations in blood pressure fluctuating from 110/60 to 160/90. Urine cleared 13 days after onset and second pyelogram found normal. Patient became comatose and died 16 days after entry.

*Differential Diagnosis.*—Kidney infection; kidney tumor.

*Ante-mortem Diagnosis.*—Indefinite.

*Treatment.*—Staphylococcus antitoxin.

*Extent of Dissection.*—Abdominal aorta on left side 3 cm. above left renal artery showed large oval-shaped defect in wall, perforation with extensive hemorrhage. Blood showed some laminations. This represented healed defect with recent rupture and hemorrhage.

*Comment.*—Anatomic diagnosis: Ruptured dissecting aneurysm of abdominal aorta with multiple septic infarcts of lung, spleen, and left kidney.

CASE 12.—Clinicopathological Cases,[30] 1942.

*Leading Symptoms.*—A 47-year-old plumber admitted complaining of steady abdominal pain of 24 hours' duration.

*Past history:* Two years ago first noted steady aching, low substernal pain, and feeling of compression which had persisted almost constantly since that time. Discomfort worse at night and relieved if walked floor. Not influenced by exertion, breathing, or swallowing. At same time, began having dull headache, became weak and tired, and developed nocturia (once). Blood pressure: 276. Twenty-four hours before admission, while eating sandwich, suddenly seized with severe steady pain beneath xiphoid, radiated through back and up between shoulder blades. Felt nauseated and vomited small amount of food. Pain increased in severity. Then he experienced severe cramp in back of right leg which remained until he stopped walking. Subxiphoid epigastric pain continued in spite of sedation.

*Physical examination:* Well-developed, well-nourished man in acute distress because of severe epigastric steady pain. Unable to lie flat on back in bed. Felt better on side, sitting, or standing erect. No pallor, cyanosis, or sweating. Lungs, clear. Heart, enlarged slightly to left; pulse, 85. Faint systolic murmur at apex. $A_2$ louder than $P_2$. No rub. Marked tenderness, without spasm, high in epigastrium; no masses. No other positive findings. Temperature, 98.8° F.; pulse, 85; respirations, 20; blood pressure, 180/110. Red blood cells, 4,500,000; hemoglobin, 100 per cent; white blood cells, 14,200—90 per cent polymorphonuclears. Urine and stools, negative. Nonprotein nitrogen, 21 mg. per 100 c.c. X-ray of abdomen: Loop of slightly dilated, air-filled small intestine in L.U.Q.; poorly defined, decreased density overlying and extending to both sides of fourth lumbar vertebra. Gall bladder x-ray, negative. Chest: Markedly tortuous aorta. Gastrointestinal series, negative. Abdominal pain persisted for 6 days requiring large amounts of morphine sulfate and then gradually subsided. Blood pressure: Both arms equal, 180/100. Urine on fourth and fifth day, 20 to 40 red blood cells. Convalescence uneventful and discharged forty-first hospital day.

Second admission, 2½ months later: Substernal pain and sense of chest constriction reappeared unrelated to exertion. Relieved with nitroglycerin.

*Physical examination:* Unchanged from first entry except heart was 2 cm. beyond midclavicular line and blood pressure, 220/140 in all extremities. Urine and blood, negative. X-ray: More definite dilatation of aortic arch. While in hospital, patient had severe 1½ hours' attack of chest pain, radiating down left arm and only partially relieved by morphine sulfate. Left radial pulse weaker than right. Blood pressure: Right arm, 170/120; left, 120/80. Next day, blood pressures about equal. Discharged twenty-fourth day.

Third admission, 9 months later: Twenty-four hours before entry, while working, experienced rapid onset of severe pain in left arm with associated mild oppression in upper sternum and pain in both sides of neck. Lasted one hour and reappeared one hour later. Not relieved by nitroglycerin. Fundi—several hemorrhages. Physical examination and laboratory data otherwise unchanged; stayed in hospital 8 days.

Fourth admission, 4 months later: Eight hours before admission, after drinking 2 glasses of beer, had sudden attack of substernal pain radiating down both arms and into neck.

*Physical examination:* Obvious pain, rapid breathing, and groaning. Heart sounds distant and poor. Blood pressure: 194/118. White blood cells, 15,000. Electrocardiogram: Biphasic instead of inverted T and $T_2$. Developed gallop rhythm, fall in systolic blood pressure to 158, pallor, sweating, and cold extremities. Showed crepitant râles and wheezes at both lung bases, tender liver with edema of sacrum and extremities, and distention of neck veins. Died suddenly on third hospital day.

*Differential Diagnosis.*—Hypertensive and coronary heart disease; acute coronary occlusion; arteriosclerosis of aorta; dissecting aneurysm; acute abdomen—first admission.

*Ante-mortem Diagnosis.*—Same as differential diagnosis.

*Treatment.*—Morphine sulfate and oxygen.

*Extent of Dissection.*—Two separate dissecting aneurysms, clearly of different ages. Older one began with horizontal tear on anterosuperior aspect of arch just distal to subclavian artery at point 9 cm. above aortic valve. This led into smooth, endothelial fat which lay within media of vessel and nearly surrounded vessel. Extended down entire aorta into right common iliac artery where it reruptured into lumen of latter vessel. Lumen of aneurysm completely endothelialized and was dotted with numerous small, bright yellow plaques of atheroma. Second independent and more recent dissecting aneurysm began with another horizontal tear 3 cm. distal to first one but on right posterior surface. In this case, aneurysm extended backward instead of forward down ascending aorta to annulus of aortic valve, thus involving mouth of both coronary aortic arteries and that of innominate artery 3 cm. above aortic valve. Outer layer had ruptured into pericardium. This aneurysm was much fresher than other and showed no endothelialization.

*Comment.*—Anatomic diagnosis: Dissecting aneurysm of aorta, old, with rupture into right iliac artery and fresh rupture into pericardium. Hemopericardium with cardiac tamponade. Compression of right renal artery with atrophy of right kidney. Surgical consultant was all for immediate transfer to surgical service and exploratory laparotomy, although he never committed himself as to precise type of surgical abdomen he expected to find.

Case 13.—Pekin and Nesbitt,[31] 1942.

*Leading Symptoms.*—A 49-year-old white man with past history of dyspnea on exertion and pain below lower sternum for past two years. Known hypertensive. Week before hospital admission, while at work, noted severe palpitation and had "pressing feeling" in apical region, which radiated down left arm. After week in bed, he experienced on day of admission, sudden pain in both sides of chest. This began in back and radiated around to abdomen and felt "like putting on a belt." Numbness in legs and within few seconds was unable to stand or walk. Numbness but no paralysis of both arms; this soon disappeared. Within hour, developed severe pain in right leg and left to lesser extent. When he entered hospital he complained of severe pain and inability to move right leg, and of less acute pain in both costovertebral angles and in epigastrium. When examined, he was lying in bed, restless, groaning with pain, and unable to move right leg. Color, good. No tracheal tug was felt. Both lungs resonant, breath sounds everywhere vesicular; no râles. Apex beat was neither seen nor felt. Left border of heart was 13 cm. and right 5 cm. from the midsternal line. Pulse rate, 40 and regular. Tones, faint. Aortic second sound equaled pulmonic second sound. No murmurs. Blood pressure: 250/150 in both arms. Abdomen, soft and slight tenderness in epigastrium. Unable to move right leg; touch sensation diminished on that side. Knee jerks and ankle jerks present and equal. No Babinski; color of both feet and

legs normal and each foot equally cool to touch. Pulsation of femoral, popliteal, and dorsalis pedis arteries in right leg were only faintly felt and of much less amplitude than in left leg. Hypodermic of morphine brought only slight relief. After two hours only slight pain in right leg persisted. Sensation to touch now absent. Blood pressure: 240/146; heart rate, 112 and regular. Color, good. Following day, patient restless due to severe pain in abdomen and groins. Legs cool, right more so than left, but no edema was noted. Color pale and cyanotic. On third hospital day, abdomen became slightly distended, size and color of legs remained unchanged. Blood pressure: 160/134 systolic; 95/100 diastolic with definite pulsus alternans perceptible at wrists. No blood pressure obtainable in legs. On fourth day, extremely restless due to pain in umbilical region, associated with nausea, vomiting of dark semidigested blood, and passage of frequent small tarry stools. That evening, skin of legs became mottled bluish-red and pain in legs and abdomen was prominent to within few minutes of death. Three electrocardiograms taken revealed no information of positive value. Bedside x-ray of chest was negative. Blood count normal; serologic tests and urine analysis, negative.

*Differential Diagnosis.*—Dissecting aneurysm; coronary occlusion with embolism to right iliac artery; mesenteric infarction.

*Ante-mortem Diagnosis.*—Dissecting aneurysm.

*Treatment.*—Sedation.

*Extent of Dissection.*—Ascending aorta and arch showed fairly smooth, yellow intima with some scattered dense, elevated areas, particularly noted about origin of large arch vessels. Between intima and media, in region of great vessels, was reddish-brown, adherent, thin laminated blood clot which extended downward for 5 cm. Remaining aorta showed yellowish streaking with longitudinal yellow calcific plaques which tended to disappear below renal artery. Entire wall from region of arch vessels to femorals and beyond internal iliacs was split posteriorly. Tear was in media. Partially organized brown, laminated blood clot, beginning 6 cm. from aortic bifurcation and into external and internal iliacs as far as they could be traced, was noted. The clot was so extensive in both iliacs as to have almost completely occluded lumen. No real tears involving intima could be seen, but there was seepage of blood through adventitia in region of arch vessels.

CASE 14.—Blain, III, Glynn, and Hiratzka,[32] 1945.

*Leading Symptoms.*—A 47-year-old colored man treated for hypertension for several years. Antisyphilitic therapy in 1925. At 9 A.M., Dec. 24, 1944, he was taken with very severe pain in left costovertebral angle and under sternum, radiating to epigastrium and to back as high as angle of left scapula. Pain, sudden in onset and constant, intensified on motion.

*Physical examination:* Well-developed, well-nourished, moderately orthopneic Negro in cold sweat. Grade 2 hypertensive retinopathy. Definite tracheal tug. Lungs clear. Blood pressure: 250/145 in each arm. Heart rate, 80 per minute; rhythm regular. Apical impulse forceful and located 11 cm. from midsternal line. Percussion dullness 4 cm. to right of midsternal line in second right interspace with no palpable pulsations in that area. Harsh systolic aortic murmur with accentuated aortic second sound heard best in second right interspace and transmitted to third and fourth left interspaces along sternal border. No diastolic murmur. Abdominal examination negative. Neurological examination negative. Marked left costovertebral angle tenderness. Roentgenogram of chest: Generalized dilatation of aorta and moderate left ventricular enlargement. Died following morning.

*Differential Diagnosis.*—Acute ureterolithiasis; myocardial infarct; dissecting aneurysm.

*Ante-mortem Diagnosis.*—Dissecting aneurysm.

*Extent of Dissection.*—Dissecting aneurysm beginning at level of left subclavian artery and extending through bifurcation down into left iliac artery 4 cm. Left renal artery involved by direct extension; right not involved. Lumen of left renal artery partially occluded.

*Comment.*—Neither kidney infarcted; arteriolar nephrosclerotic kidneys; no evidence of syphilitic aortitis; no intimal rupture.

CASE 15.—Blain, III, Glynn, and Hiratzka,[32] 1945.

*Leading Symptoms.*—A 61-year-old Negro man, while sitting in theater, was taken with excruciating pain in right costovertebral area, radiating down toward groin and medially toward midline of chest. No nausea or vomiting. History of high blood pressure.

*Physical examination:* Acutely ill, obese man, in severe pain, covered with sweat. Slightly orthopneic. Blood pressure: 270/150; pulse, 100; temperature, 99.2° F. Heart enlarged to left. Systolic blow at apex and at base. Moist râles at both lung bases. Chest x-ray: Minimal congestion of lung fields and broadening of aorta. Flat plate of abdomen, negative. Urine, 4 plus albumin; 30 to 40 white blood cells; few granular casts and innumerable red cells. Blood count: hemoglobin, 13 Gm., white blood cells, 14,500; 95 per cent neutrophiles. Electrocardiogram: Left axis deviation and myocardial damage. Some ecchymotic areas were noted on skin of abdomen and flank. Serology positive. Blood urea, 124. Urea clearance, 20 per cent of normal. For first three days, pain could not be adequately controlled with morphia. Later, pain subsided. Within week, patient had sudden severe pain in chest and fell dead.

*Differential Diagnosis.*—Hypertensive heart disease with decompensation; right nephrolithiasis; dissecting aneurysm involving right renal artery; syphilitic aneurysm.

*Ante-mortem Diagnosis.*—Hypertensive cardiovascular disease: Syphilitic aneurysm of arch; renal infarction and/or dissecting aneurysm of abdominal aorta with renal artery involved.

*Treatment.*—Morphine sulfate.

*Extent of Dissection.*—Aorta—arteriosclerosis with dissecting aneurysm extending from base of left subclavian artery to iliacs. Dissection was filled with organized blood clot and involved proximal 3 cm. of right renal artery. Proximal 16 cm. of dissecting aneurysm exhibited organized blood clot 1.5 cm. in thickness. On external surface of aorta halfway around segment of blood clot was nodular elevation 3.5 cm. in diameter which had irregular perforation. Through this, blood (1,700 c.c.) had escaped to produce terminal hemothorax.

*Comment.*—No syphilitic aortitis.

## DISCUSSION

The etiology, pathogenesis, clinical manifestations, and laboratory findings have been reviewed previously,[3] but a brief summary may help to recall this entity.

The etiology of dissecting aneurysm is unknown, and many factors[4, 5] have been mentioned; however, arteriosclerosis and hypertension are frequent associated findings, and would seem to be at least predisposing factors. Syphilis plays no part. The pathologic lesion is one of medial degeneration with subsequent hemorrhage and dissection between the middle and outer one-third of the aortic media. One usually finds an internal rupture present in the ascending portion of the aorta, just above the aortic valve,[6] but this is not necessary for dissection to occur, and many cases have been reported where no intimal tear was present.[1, 7, 8]

Clinically, a past history of hypertension is common, but a history of straining or strenuous exercise is not as frequent as generally assumed.[9] The onset is sudden, with excruciating pain, described as "knife-like" or "tearing," which is often localized in the lower substernal region or upper midepigastrium. The pain frequently radiates into the neck, interscapular region, or down the abdomen into the legs, but seldom down the arms unless the brachial arteries are occluded. With the initial pain, the patient commonly experiences faintness, dyspnea, nausea, and vomiting. The patient appears in a state of

collapse, with pallor, profuse diaphoresis, anxiety, and restlessness. Surprisingly, the blood pressure is often normal, or high, and the pulse of good quality with normal rate. The heart is enlarged due to previous hypertension, and systolic and diastolic murmurs may be audible over the precordium. However, the presence of a diastolic murmur over the aortic area, which was not audible previously, is important and may indicate that cardiac tamponade is pending.[10] The dissection may extend into the aortic branches, partially or completely occluding them, producing hemiplegia (carotid arteries); unequal peripheral pulses, and blood pressure, plus cold, cyanotic, flaccid extremities (brachial, femoral or iliac arteries); hematuria and anuria (renal arteries); intestinal obstruction and melena (mesenteric arteries). Roentgen studies are valuable and reveal widening of the aortic arch with dilatation of the descending aorta, cardiac enlargement, and pleural fluid, if present.

The eventual outcome in 95 per cent of the cases is external rupture of the aneurysm, 70 per cent into the pericardial cavity, 20 per cent into the left pleural cavity, and 5 per cent into the mediastinum.[11] The external rupture usually occurs in two or three days, but may not occur for months or years. Occasionally, the aneurysm may rupture back into the aortic lumen with the formation of a "double-barreled" aorta. Ninety per cent of the patients with dissecting aneurysm die during the first attack.

From this brief discussion, one can see where familiarity with the disease would have made one suspect this entity in the surgical cases (Cases 3, 4, 5, 6, 9, 15, 16, 19, and 20) and perhaps prevented an operation.

In eighteen of the 278 cases reported (6.5 per cent), surgical lesions were recorded as incidental findings at autopsy. These included such lesions as chronic cholecystitis with cholelithiasis, acute pancreatitis, carcinoma of the bronchus and stomach, paraganglioma, and diverticulosis of the sigmoid.

In reviewing the cases where dissecting aneurysm was considered in the differential diagnosis, one can readily appreciate the number of surgical diseases this entity might simulate (Tables I and II). However, if the few manifestations already mentioned are kept in mind, the diagnosis will not prove difficult.

As yet there is no method for correcting this disease and the treatment consists of rest, sedation, and analgesics.

## SUMMARY

Dissecting aortic aneurysms cause protean symptoms among which the symptoms of "acute abdomen" and other surgical diseases are more prominent than has been heretofore emphasized. A survey of the American literature for the past eleven years has revealed that in 12.9 per cent of dissecting aneurysms a mistaken diagnosis of some surgical disease was made or considered. In one-half of the cases diagnosed as surgical disease the patients were subjected to futile operative intervention. We feel that this evidence is sufficient to warrant the surgeon's familiarity with the symptoms and other features of dissecting aortic aneurysms lest he add to the list of useless and perhaps lethal operations performed when this disease is encountered.

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# Correspondence

## A Note on the Halsted Operation for Inguinal Hernia

*To the Editor:*

The August, 1948, issue of SURGERY contained an article by Farris, Ettinger, and Weinberg[1] entitled "The Hernia Problem, With Reference to a Modification of the McVay Technique." In speaking of the Halsted operation, the article carries as a footnote the following statement: "Contrary to general opinion, the Halsted operation employs the principle of suture of the internal oblique and conjoined tendon to the shelving edge of Poupart's ligament without anterior transposition of the cord."

The authors are in error in making this statement. As the influence of the written word is very far-reaching, I wish to point out just what the error is and a possible reason for its being made.

Halsted[2] and Bassini[3] developed their operations simultaneously and entirely independently of each other, and published reports of their operations at approximately the same time (1889-1890). (Bassini did report his operation earlier but the reports were published in little-read Italian journals[4, 5] and therefore did not reach the attention of the medical public.) Both authors sutured the conjoined tendon and internal oblique muscle to Poupart's ligament and their operations differed principally in their methods of handling the cord. Bassini transplanted the cord between the internal oblique muscle and the aponeurosis of the external oblique. Halsted transplanted the cord external to the aponeurosis of the external oblique in the subcutaneous position. As a matter of fact, Halsted[6] did at the very outset test the relative value of cord nontransplantation, and in some cases permitted the entire cord to lie undislocated and altogether undisturbed in its bed. However, this work was not published until after Ferguson published reports of his operation in which the cord was not transplanted. Halsted also in his original operation resected most of the veins of the cord, but later abandoned this procedure because in some cases atrophy of the testicle resulted from it.

Later on (1903), Halsted[6] abandoned his original operation and started using the Ferguson-Andrews operation, which he considered better than his own original procedure. (Actually, as will be shown later, he developed this method independently of Ferguson and Andrews.) He always had an open and an independent mind and was quick to adopt the work of other people if he thought it better than his own. As Halsted was the leading exponent of hernia surgery in America, his newly adopted operation began to be called, in places outside of Baltimore, the "Hopkins" operation and also the "Halsted II" operation. Both of these names are misnomers because credit for the operation should go to Ferguson[7] of Chicago, who was the first of the post-1890 workers in the field of hernia surgery to adopt and publish the method of leaving the cord in its bed, not transplanting it at all; and to Andrews,[8] also of Chicago, who was the first American to adopt and publish the principle of the imbrication of the aponeurosis of the external oblique. Lucas-Championnière[9] in France had utilized the principle of the imbrication of the aponeurosis of the external oblique and had published his work three years before Andrews' publication appeared, so strictly speaking the Ferguson-Andrews operation should be called the Ferguson-Lucas Championnière operation. However, the former name has become so ingrained in the nomenclature of American surgeons as to be almost impossible to dislodge.

It is easy, then, I think to understand why the authors previously mentioned fell into the error of thinking that the cord was not transplanted in the Halsted operation. Had they gone back to his original article[2] his method of handling the cord would have been clear to them. The article of Halsted which they quote, namely that published in 1893,[10] still carries the account of his original operation and describes the subcutaneous transplantation

of the cord. His 1903 article[6] gives an account of the operation which he was doing at that time, and which was really the Ferguson-Andrews operation. This operation was a gradual development on the part of Halsted and he[6] had actually employed both of the distinguishing characteristics of it (the nontransplantation of the cord and the imbrication of the aponeurosis of the external oblique) before either Ferguson's or Andrews' articles were published, as related in his 1903 article. This was another case of different workers developing the same idea simultaneously. However, the credit goes to the first in print and therefore I believe that "Ferguson-Andrews"* is a more proper designation of the operation adopted by Halsted and independently developed by him, than the term "Hopkins" operation, or "Halsted II" operation. Halsted was a very careful observer, a meticulous operator, and a painstaking investigator. It was probably due to all of these characteristics that he delayed publishing the operation which might very properly have been called the Halsted II operation had not the salient points of it been previously published by other workers. Halsted, I am sure, would be the last to wish that any credit be given him that belonged even partly to someone else.

There is constant evidence in surgical writings, addresses, and conversations that there has been a good deal of confusion with regard to the Halsted operation. A recital of the facts in no way detracts from the great surgeon that he was. On the contrary, the more one delves into his writings, the more one is arrested by his originality and scientific genius.

—*Amos R. Koontz, M.D.*
Baltimore, Md.

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*While Lucas-Championniere[9] was the first to imbricate the aponeuroesis of the external oblique, Andrews stressed it more and used a more elaborate imbrication, placing the cord between the two flaps of the aponeurosis—a method now little used. There is much to be said against using surgeons' names to designate operations, but certain operations have become so well known as identified with certain surgeons that it is difficult to change them. The Ferguson-Andrews operation is one of them. Those interested in the history of the operation for hernia, though, will want to bear in mind the part played by others in developing this operation.

# Book Reviews

**Fundamentals of Clinical Neurology.** By H. Houston Merritt, M.D., Professor of Clinical Neurology and Chief of Division of Neuropsychiatry; Fred A. Mettler, M.D., Ph.D., Associate Professor of Anatomy, and Tracy Jackson Putnam, M.D., Professor of Neurology and Neurological Surgery, College of Physicians and Surgeons, Columbia University. Pp. 289, with 94 illustrations. York, Pa., 1947, The Blakiston Company.

This small treatise on clinical neurology is obviously written around Mettler's *Neuroanatomy* and whatever claim to distinction it may have among the many current textbooks on clinical neurology is largely due to the excerpts, especially the anatomic illustrations, from this well-illustrated neuroanatomy.

The final chapter consisting of twenty-six pages on the spinal fluid is a very good chapter. It is relatively large in relation to the other clinical discussions in the book and no doubt reflects Merritt's interest in the spinal fluid as a result of his book with Fremont Smith on this subject.

In the introduction the book is said to be primarily for the general practitioner of medicine. It is not the most easy reading he could find on the subject, but the neuroanatomy illustrations will be helpful to him.

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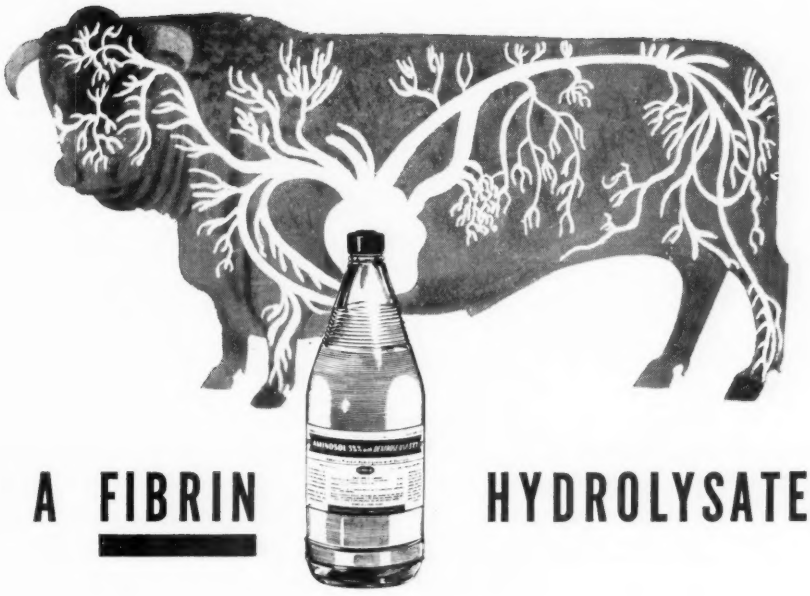
**Retropubic Urinary Surgery.** By Terence Millin, M.D., Surgeon, All Saints Hospital for Genito-Urinary Diseases, London. Pp. 208, with 163 illustrations. Baltimore, 1947, Williams & Wilkins Company.

This is a good book, well illustrated, which will be read eagerly by anyone interested in prostatic surgery. The experienced prostatectomist will realize that the author is straining just a little to put his ideas across, and will be inclined therefore to discount some of his rather unkind remarks about approaches to the prostate other than his own, and to chortle over the fact that "operative difficulties" require but two pages. It is annoying to find an index so fragmentary as to contain no mention of such minor matters as mortality, hospital stay, etc.

There are interesting and rewarding chapters on the pathology of the prostate gland, surgical anatomy, pre- and postoperative care, retropubic enucleation of the prostate, retropubic operations for prostatic carcinoma, calculi, fibrosis, and abscess, for carcinoma of the bladder, and for incontinence in the female, and on a perineal operation for urinary incontinence in the male. This consists of wrapping Lowsley's ribbon catgut many times around the bulbous urethra in the male and tying it tight. It sounds good, although your reviewer has, probably owing entirely to his own ineptitude, produced four strictures with it.

The operations are so well described and illustrated that the reader, given sufficient skill, should have no difficulty in reproducing them. Owing to the deficiencies of the index, I cannot find the author's figures on mortality, but it seems to me that it was about 4.5 per cent in 375 cases. This will undoubtedly seem pretty high to the members "of a leading middle western transurethral clinic" mentioned with some annoyance by the author, but is very good when everything is considered (drug shortages in Europe, operations in many hospitals in many cities, etc.).

Chief advantages claimed for retropubic enucleation of the prostate are good exposure of prostatic fossa, comfortable convalescence, early healing of the wound, and short period of hospitalization. It will take some years for this method to find its proper place among the older and better established methods.



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Published by THE C. V. MOSBY COMPANY, 3207 Washington Blvd., St. Louis 3, U.S.A.

Great Britain Agents: Henry Kimpton, Ltd., 26 Bloomsbury Way, London, W.C.1.
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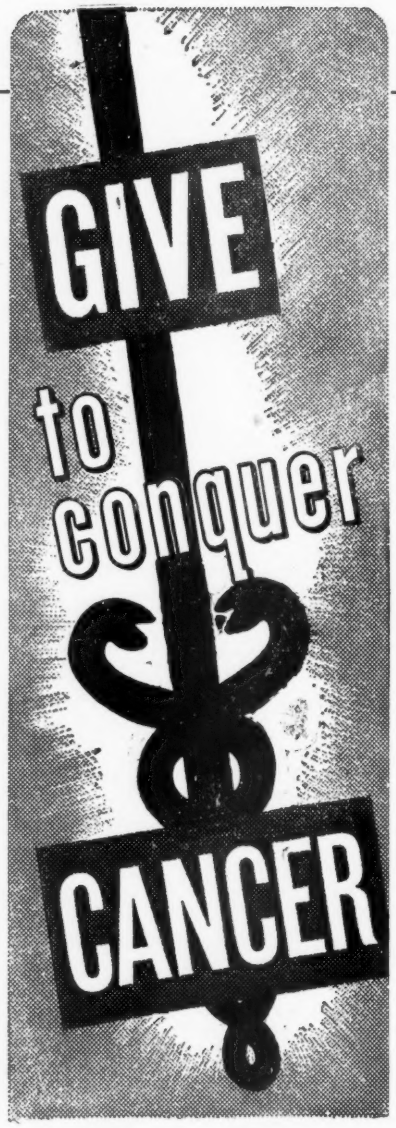


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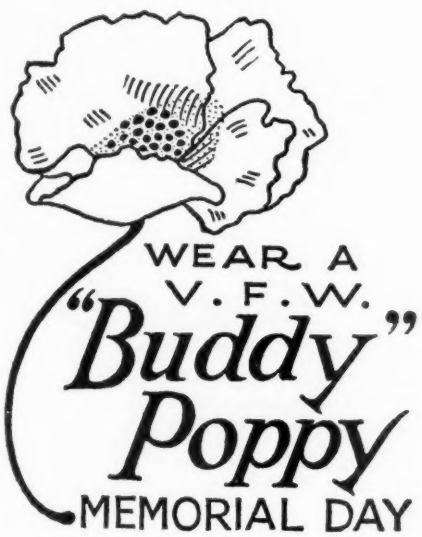



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